ACTA RADIOLOGICA

EDITA PER SOCIETATES RADIOLOGICAS DANIÆ, FENNIÆ, HOLLANDIÆ, NORVEGIÆ ET SUECIÆ

VOL. II FASC. 4—5 18: IX 1923 N:o 8—9

RADIOLOGICAL TREATMENT OF CHRONIC TONSILLITIS

Introductory Address at the 3rd Congress of the Northern Association for Medical Radiology in Stockholm June 18th and 19th 1923

by

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(Tabulæ IX—XIV.)

Introduction.

When the »Northern Association for Medical Radiology» in September 1921 laid upon me the task of introducing the discussion on the radiological treatment of chronic tonsillitis, I had then already for two years been greatly interested in that form of treatment.

NOGIER and RÉGAUD had already in 1913 described a method for roentgentreating hypertrophic tonsils, and in 1920—1921 some American roentgenologists with WITHERBEE at their head published a series of papers on the roentgen treatment of chronic tonsillitis. But any exhaustive work on the radium treatment of this complaint had not been published.

When planning my preparatory work for this introductory address I had thus two ways by which to proceed. One alternative was to try to apportion the material, which might eventually be at my disposal, to the different radiological methods of treatment and then to give a critical and comparative account of the results obtained by the different methods. The other alternative was to treat the whole of the material by one, as far as possible, uniform method of treatment and then give a detailed statement of the advantages and drawbacks of this method.

For two reasons I chose the last alternative so as not to split up the work on several lines.

Firstly, because the question would no doubt be investigated more thoroughly if I tried in this way to deepen and to concentrate my own practical experience to a smaller sphere and thereby gained a settled personal opinion of the different problems concerning the treatment. Secondly, I considered the main point to be whether and to what extent a radiological treatment of the chronic tonsillitis would be possible and would give a good clinical result, and not whether a radium or a roentgen treatment was to be preferred.

By collating the results that other investigators had obtained by methods which they have elaborated for the roentgen treatment of the chronic tonsillitis, the other side of the question might also be elucidated.

For this reason, then, I have treated my material uniformly on a common principle, from which I have only occasionally digressed.

Short history of the anatomy, embryology, physiology, pathology and physiological involution of tonsillar tissue.

I. During the latter part of the 19th century some classical investigations were made by the great anatomists KÖLLIKER, BILLROTH, HENLE, BRÜCKE and others in order to analyze the anatomy of the lymphoid tissue.

This tissue consists of a ground substance of mesoblastic cells which form a mesh-work, a reticulum, extending also between the walls of the lymphatic vessels. The meshes of this reticulum are closely packed with lymphocytes, which have the characteristic lymphocyte nucleus with a close reticulum of pachychromatin and a distinct nuclear membrane. Around the nucleus there is a relatively large zone of protoplasm. This lymphocytic portion of the lymphoid tissue fills up the meshes, and covers generally the cells of the reticulum. It may, however, differentiate into rounded formations of closely packed lymphocytes, s. c. germinal follicles or, better, secondary follicles, which formations reach the size of a pin's head and can often be observed macroscopically. Within such a follicle one finds often a clear central portion in which the meshes are wider and the lymphocytic elements larger than those in the periphery, where lymphocytes of an ordinary appearance are lying closely packed in a zone round the clear centre. These formations were discovered by FLEMMING in 1885, and he and his disciples have tried in a number of investigations to ascertain the nature of their function.

The lymphoid tissue of the throat is concentrated to the so-called ring of WALDEYER. This ring consists partly of a diffuse infiltration of the pharyngeal mucous membrane by lymphoid tissue and partly of portions of this lymphoid tissue collected to form true organs. Thus, one distinguishes between the following groups of lymphoid tissue:

1) the palatine tonsils, situated in the fauces on either side of the pharynx;

2) the lingual tonsil, forming a diffuse infiltration by lymphoid tissue on the base of the tongue, the infiltration concentrating very often so as to form macroscopically observable tonsillar formations;

3) the inconstant accumulation of lymphoid tissue around the Eustachian tube;

4) the pharyngeal tonsil, situated in the nasopharynx where the septum nasi passes on to the posterior wall of the pharynx;

5) LEVINSTEIN's inconstant tonsils in the lateral wall of the pharynx.

MOLLIER has characterized the tonsils as lymphoepithelial organs, as he considers that their development and anatomy as well as their function is due to a co-operation of epithelium and lymphatic tissue.

II. The tonsils begin their formation during the fourth month of fetal life as involutions of the epithelium, around which lymphoid tissue accumulates rather irregularly. The tonsils reach their full development during the first year after birth, the simple epithelial involutions growing into branched crypts which penetrate deep down into the tissue. The lymphocytes, which have before been rather irregularly collected, begin at the same time to become differentiated into the above-mentioned secondary follicles with a clear centre and dark peripheral zone.

A congenital hyperplasia of the lymphoid tissue as a part symptom to the so-called Status lymphaticus is not met with. The whole theory of Status lymphaticus has been shaken to its very foundations by the investigations of LUBARSCH, and HELLMAN has also pointed out the unjustifiable in advancing this idea as a constitutional anomaly.

III. Quite a number of different hypotheses have been advanced as to the function of the tonsils. KÖLLIKER considered, for instance, that the tonsils in the pharynx only served as padding in the same manner as the adipose tissue. BOSWORTH and others were of the opinion that the lymphoid tissue had something to do with the digestion and served as secretory organs. HARRISON believed that they are in some way in the service of the internal secretion.

The modern theories of the physiology of the lymphoid tissue ascribes to it the following three functions:

1) formation of lymphocytes;

2) a filtering off of foreign bodies that get into the organism;

3) destruction of toxins, bacteria and other injurious matters.

The first function was pointed out by FLEMMING and his disciples already about 1880. They found that there are numerous mitotic figures in the follicles, and especially in their centres, and assigned the primary seat of formation of the lymphocytes to the clear centres, which they therefore called germinal centres or Keimzentra.

The second function was also verified at an early period by the demonstration of the filtering capacity of the mesh-work of reticulum cells that stretches between the walls of the lymph vessels in the lymphoid tissue, and HELLMAN has proved this conclusively by his investigations concerning the behaviour of the lymphoid tissue in absorbing foreign particles and tumour cells.

The third function is, however, very much debated. Here one meets with two greatly diverging views, of which one absolutely denies that the tonsils have to perform any protective function at all, whereas the other maintains exactly the reverse. The representatives of the different views have published quite a number of histological and pathologic-anatomical investigations, experimental as well as clinical, the results of which are in direct opposition to one another.

Several clinicians profess the nihilistic view. They maintain that the tonsillar tissue in the pharynx as well as the adenoid tissue of the vermiform appendix is only an embryonic remainder, and thus functionless. Owing to the anatomical structure with numerous crypts, which constitute a splendid place for pathogenic bacteria to live in, and by the loose condition of the epithelium with the numerous transient lymphocytes, physiological wounds appear which are excellent points of entrance for infections. The adenoid tissue in the pharynx, and especially its accumulations into large organs, constitute a perpetual menace to the organism and should be completely removed as early as possible.

It is on the basis of this theory of the tonsils being particularly dangerous organs for the body, that the tonsillectomy has gained ground.

Again, the authors favouring the view of the protective function of the tonsillar tissue are divided up into different schools. The most prominent directions are:

1) The phagocyte theory;

2) The protective theory of BRIEGER-GOERKE and

3) HELLMAN's theory of the secondary follicles as centres of reaction against invading injurious matters.

According to the first theory, the chief protection would be afforded by the activity of the phagocytes. As these, however, do not come up in number to the lymphocytes which dominate within the lymphoid tissue, and the lymphocytes not having any direct phagocytotic power, this theory has won only a small number of followers.

A flow of lymph is, according to the theory of BRIEGER-GOERKE, taking place from within the tonsil towards its surface, and by this flow foreign bodies and bacteria would be prevented quite mechanically from invading the tonsillar tissue, the lymph having at the same time a specific bactericide power.

The theory of HELLMAN is founded on a research work of long duration upon the lymphoid tissue. Thus, he has inquired into the amount of this tissue in rabbits of different ages and its state during physiological and pathological conditions by a series of extremely close and critical investigations, through which he has arrived at a very acceptable theory regarding the function of this tissue.

The amount of lymphoid tissue in rabbits at different post-fetal ages shows two maxima. The first maximum is at the age of five months, i. e. at the time of puberty. The second maximum is at the age of ten months. HELLMAN considers these maxima to be indicative of the lymphoid tissue having reached its highest condition at these periods. The top of the weight curve at the age of five months is given by all lymphoid tissue, but especially by the more deeply embedded tissue, and coincides, as to time, with the period of the greatest abundance of the lymphocytes in the blood. This top is a manifestation of the intense function of the lymphoid tissue at that time in producing lymphocytes.

The second top on the weight curve of the lymphoid tissue only appears in the case of the superficial lymphoid tissue and becomes particularly prominent as far as the weight of the secondary follicles of the rabbit's tonsil are concerned, because this is largest in animals at the age of ten months, both with regard to its absolute and relative proportion to the ground tissue. HELLMAN associates this apex with the function of the superficial tissue to guard the organism against invading foreign bodies and matters.

The increase in weight being largely due to an increase in the number of the secondary follicles, he considers them to be the anatomical basis for the protective function of the lymphoid tissue.

HELLMAN is of the opinion that the lymphoid tissue is the place

where the lymphocytes are produced, but in opposition to FLEMMING and his disciples he does not assign this production to the secondary follicles but to the ground substance of the lymphoid tissue. And he advances many weighty objections against the validity of the theory of FLEMMING.

The secondary follicles are, thus, a functional differentiation, centres of reaction, indicating that the tissue is active in rendering the bacteria or other toxins harmless.

By this theory of the function of the lymphoid tissue it has been possible for HELLMAN to explain many phenomena which have not been explained at all before, and he has also been able to shed light upon quite a number of clinical problems. Concerning the interesting details I must refer my readers to the original publications of HELLMAN, as the space here does not allow of my entering further upon them.

IV. The simplest pathological change, consisting of a hypertrophy of the tonsillar tissue, might perhaps, thus, be due to an inferior quality of the tissue, caused by some factor or other (nutritive disturbances or the like), which inferiority the tissue then tries to counterbalance by an increase in quantity.

Because of their loose surface and deep crypts these hypertrophic tonsils are very liable to infections, and if these increase in intensity, the task of repelling the infection becomes too great and an inflammation sets in, followed by hyperemia, edema, accumulations of leucocytes, macrophages and partial necroses. The protective function of the tonsil is further weakened by this inflammation, and a stage is finally reached, when the tonsil is overcome by the infection time after time and gradually passes into a chronic state of inflammation, when it can no longer fulfil its function.

This function must then be undertaken by some lymphoid tissue situated more deeply, the lymphatic glands of the neck. A tonsil thus disturbed in its function ought either to be removed or to be given a chance to regain its functional power by some form or other of local or general treatment.

Foreign irritants as bacteria and other poisonous matters find their way daily into the organism, and the lymphoid apparatus must therefore be working continuously against these invading irritants.

If the secondary follicles are centres of reaction against the invading injurious matters, then they must be in a perpetual state of transformation, consisting of progression and regression. Consequently one should be able to find centres of reaction with very varied appearances in the tonsillar tissue and particularly in the portions chronically inflamed.

For quite some time it has been a well-known fact that these centres of reaction present a characteristic appearance, for instance in diphteria, in so far as the lymphocytes become broken down in the centre, which is then occupied by large cells with a clear nucleus. These changes have been considered to be focal necroses caused by toxins of diphteria (RIBBERT, BIZZOZERO, COUNCILMAN, MALLORY and PEARCE). HELLMAN has described similar changes in a number of patients suffering from acute poliomyelitis, tetanus, congenital lues, purulent meningitis etc., and he maintains that these changes are indicative of the function of the follicles to overcome the toxins.

By an examination of 200 tonsils in patients suffering from chronic tonsillitis HEIBERG shows that secondary follicles of very varied appearances may be found in such cases. One type, which HEIBERG considers to be a well-developed follicle, exhibits well-stained cells in the centre, lying relatively close together. The chromatin shows the typical arrangement with a beautiful granular mesh-work, and the nuclear membrane has a very distinct appearance. More or less numerous mitoses may be found. As a normal feature of these secondary follicles he mentions also some regularly arranged phagocytes. As distinct from this he describes a type of secondary follicles with badly-stained irregular nuclei and badly-preserved phagocytes.

He adopts the theory of HELLMAN and supposes these last centres of reaction to be dissolving centres which have performed their protective function.

Besides these varied pictures of the secondary follicles, which pictures are indicative of the function of the follicles to guard against invading irritants, one notices very abundant infiltrations by lymphocytes in the epithelium of the chronically changed tonsils, the infiltrations resulting in some places almost in a reticulation of the epithelium. Further there are numerous foci of leucocytes in the lymphoid tissue as well as in the epithelium and the surrounding connective tissue. Large groups of plasma cells and eosinophils are frequently occurring.

V. Numerous investigations have been carried out as to the development of the human tonsillar tissue at different ages.

Although the views on this subject are very diverging, most authors consider, nevertheless, that the tonsillar tissue has its greatest development during childhood up to the age of puberty. At this age there is a pause in the development, and after the age of about 25—30 years an involution takes place, which results in the tonsillar tissue having almost completely disappeared at the age of 50.

Only a few solitary crypts are then remaining with irregular

clusters of lymphocytes in the surrounding tissue, which condition greatly resembles the embryonic stage.

The physiological age-atrophy has been described in detail by GOERKE and LEVINSTEIN.

This atrophy begins, according to them, with the disappearance of the mitoses in the germinal centres, whereupon the clear central portion in the follicles gradually disappears. Finally, the follicles are reduced in number and size and there remains only a diffuse accumulation of lymphocytes, and they, too, disappear by degrees.

Short history of the radium treatment of chronic tonsillitis.

GUNNAR HOLMGREN was one of the first who pointed out the great possibilities of the radium therapeutics in the treatment of chronic hyperplasia of the tonsillar tissue. In a lecture in the Swedish Medical Society in October 1917, he reported on the results obtained in some cases of defective hearing which would not yield to the ordinary methods of otological treatment. The defect was caused by a stenosis of the Eustchian tube due to hyperplasia, and in one case due to malignant degeneration of the lymphoid tissue surrounding the Eustachian tube. He pointed particularly to the extraordinarily quick result. The patients had got a normal hearing already on the day after the treatment, and the improvement was permanent. The lecture is published in Acta Otolaryngologica 1918, pag. 201. Simultaneously with HOLMGREN, however, STURE BERGGREN had employed radium treatment for chronic salpingo-tympanitis caused by stenosis of the Eustachian tube which, again, was due to hyperplasia of the lymphoid tissue of the nasopharynx. He, too, affirms the almost immediate effect of the radium treatment.

Numerous cases of malignant tumours originated from the ring of Waldeyer are mentioned in the literature, whilst the inflammatory processes seem to have attracted less interest.

In 1921 WILLIAMS published a paper in which he describes an original technic for the treatment of tonsillar hypertrophy, and since then SIMPSON, WELLS, OSGOOD, WITHERS, LANE and others have published reports on series of radium-treated cases of chronic tonsillitis.

The characteristic feature of the American technic is, that the treatment is given with low-filtered containers, whereby a large portion, up to 50 %, of the β-radiation passes through the filter. Different methods are employed by the Americans, and I shall here only give a short account of some of these methods.

1) WILLIAMS employs 26 m. gr. radium in a »flat container», filtered through 0,83 mm. Al.; time of treatment 15 min.; the treatment being repeated 2—3 times. SIMPSON uses the same low-filtered radiation with 30 m. gr. radium in a »flat dermatological applicator with a single layer of rubber-tissue». He treats the tonsil for one hour, giving thus a dosage of 30 m.gr.-hours. By this dosage of 30 m.gr.-hours radiumelement, with 50 % of the β-radiation, he gets a »mild reaction which turns the part white». SIMPSON combines, however, this treatment with external roentgen treatment. ROBINSON uses an unscreened 30 m.gr. plaque for one hour.

2) WELLS inserts into the tonsils 3 needles, each containing 10 m.gr. radium, time of treatment 2 hours, the dosage being thus 60 m.gr.-hours radiumelement. He does not state the thickness of the filter.

3) WITHERS recommends implanting of »bare tubes» having a strength of 0,2—0,3 millicuries and uses one tube for every c. c. of tonsillar tissue.

4) ROBINSON holds, by means of the Cameron adjustable radium-applicator directly against the tonsil 4 tubes, each containing, 12,5 m.gr. radiumelement, filtered through 0,4 mm. steel, 1 mm. brass and a layer of »rubber-tissue», the dosage being 50 m.gr.-hours. In some cases he implants two 12,5 m.gr. radium needles into the centre of the tonsil for 2—4 hours.

They all emphasize the importance of the β-rays, as these are said to have an immediate bactericidal effect and the lymphoid tissue is said to be particularly susceptible to this kind of radiation. Most of the radiation is then absorbed by the superficial layers of the tonsillar tissue.

The result of the treatment is, according to all of them, that the tonsils decrease in size and the fissured surface becomes smooth and even. Owing to the shrivelling of the tonsil the crypts become widened and completely drained, whereby their contents disappear. The patients are relieved of their recurring attacks of angina. Soft, hypertrophic tonsils of children and soft tonsils of adults are most susceptible to the treatment, whereas the small fibrous tonsils of adults are said to be less susceptible.

The majority of the authors have made bacteriological examinations and have then found that the hemolytic streptococci disappear during the 6th or 8th week after the treatment and are then entirely absent. This effect on the bacteria has been attributed partly to the bactericidal power of the β-radiation and partly to the improved »drainage» of the crypts.

A great many authors have used external roentgen radiation as an auxiliary treatment, according to the principles laid down by WITHERBEE.

Short history of the roentgen treatment of chronic tonsillitis.

As early as in 1913 NOGIER and RÉGAUD published a method for the roentgen treatment of chronic tonsillitis. They employed comparatively small dosages and obtained very favourable results, especially in the case of soft hyperplastic tonsils; whereas the fibrous tonsils proved to be more resistant to the treatment. This information seems, however, to have attracted comparatively little attention.

Only in 1921 are any further reports with regard to this question beginning to make their appearance in the American literature. WITHERBEE has laid down the principles of the roentgen treatment of the chronic tonsillitis by a series of classical investigations. He and his followers, MURPHY, CRAIG, HUSSEY, STURN, LAFFERTY, PHILLIPS, PACINI, VAN ALLEN, CHANDLERS, SIMPSON, DEMPSTER, ROBINSON, OSGOOD and others, have published a great number of reports on their experiences of the treatment and its results.

Every one of them employs principally the same technic, which has been indicated by WITHERBEE. The patient lies face downward, his head turned to one side. The central ray is focussed on a point immediately behind the angle of the jaw and is directed against the two tonsils and the nasopharynx. The field of incidence measures about $7\,^1/_2 \times 6\,^1/_2$ cm., and the surrounding parts are carefully protected by means of lead covers. WITHERBEE recommends a third area of treatment from the back of the head. Spark-gap 17,5 cm. Milliampères 5. Distance 25 cm. Time of treatment 4 min. Filter 3 mm. Al.

Both sides are treated each time and the treatments are repeated within two weeks of each other until a result is obtained, i. e. until the tonsils have decreased in size and the pathogenic bacteria disappeared. As a rule 6—8 up to 14 series of treatment are needed. The dosage employed »gives one skin-unit of filtered ray, which corresponds to $^1/_2$ skin-unit unfiltered in effect on the skin».

WITHERBEE employed, at first, stronger and more concentrated dosages, but he now states that he gets a better effect by the weaker and more prolonged treatment.

All these authors have treated a great number of cases, and WITHERBEE has a practical experience of 500 cases which he has treated during 1920 and 1921. The results of the treatment are

very favourable. The tonsils decrease in size without any visible reaction either on the skin or on the mucous membranes and without any accompanying subjective troubles whatever. They are, however, not completely atrophied, as the treatment is discontinued comparatively early. During the shrivelling process which sets in already in the 2nd and 3rd week after the commencement of the treatment, one notices how the crypts get smaller and the draining becomes more complete, whereby the contents of the crypts disappear. He emphazies particularly that it is not necessary to carry the diminution in size further than to the point where the pathological symptoms disappear, as there is no reason to continue the treatment after the patient having become clinically healthy and, further, he points out that also the fibrous tonsils are susceptible to roentgen treatment.

Most authors have made very close bacteriological investigations during the course of treatment. Out of 36 cases treated by WITHERBEE, all of whom had numerous colonies of hemolytic streptococci at the commencement of the treatment, 32 cases have been entirely rid of bacteria after 4 weeks. This result agrees very well with the observations made by HICKEY who has roentgen treated diphteria carriers and got 80 % of the cases absolutely free from symptoms 2—4 days after the commencement of the treatment.

This effect on the bacteria is largely ascribed to the improved drainage of the crypts.

Finally, WITHERBEE states that the method is not only safe and gives a permanent result, but it also removes the focal infection more certainly and more completely than any other method.

VAN ALLEN has made a post-examination of 50 patients treated with roentgen for tuberculous lymphoma, and he then found a distinct effect on the tonsils of the patients. They had all been under observation for at least 3 years after the end of the treatment and had exhibited numerous attacks of angina in their anamneses. Eighty per cent of them had not had a single attack of angina after the treatment, the tonsils had become small and the glands had also decreased in size. The 20 % who had attacks of angina, declared that these attacks had now been much slighter than before the treatment.

The American authors all emphasize that the radiumtreatment as well as the roentgentreatment influences favourably the secondary symptoms, which may be associated with chronic tonsillitis, for instance neuralgic pains, recurring rheumatic arthritis, chorea, lymphadenitis, tuberculous lymphomata and symptoms from the kidneys.

Radiumtreatement of chronic tonsillitis.

Technic of application.

I. Several authors have constructed various apparatuses for the application of radium in the oral cavity and the pharynx. ALBANUS and STICKER have, thus, constructed some applicators for fixing the radium directly against the surface to be treated. Tongs-resembling instruments have been described by BERVEN and BROEMAN, the latter of whom uses the hemostat of Corwin & Wilder with a contrivance for the fixation of the radium preparations. Some other American authors make the patients themselves hold the radium, which is fixed on a holder, against the tonsillar surface. HIRSCH describes an arrangement in which the holder is fixed against the teeth by means of a plastic mass. EDLING and SCHÉELE have also constructed special apparatuses for this kind of application, where the fixation is produced by means of a plastic mass against the teeth. A good instrument for application must answer the following requirements:

1) The fixation must be firmly secure in such a manner that the preparations are not displaced if the patient swallows coughs, vomits or the like.

2) A steady, firm pressure must be applied on the tonsil. This pressure is endured quite well by the patients, whereas small displacements of the preparations against the mucous membrane or variations of pressure will easily provoke vomiting movements.

3) The application must be of as little inconvenience as possible to the patient. The free movements of the tongue should not be impeded.

4) One must always have an opportunity, while the treatment is in progress, of seeing that the preparation is not displaced.

5) The applicator must be easy to clean.

The application arrangement employed by me in the treatment of the majority of my cases is an improvement of the tongs which I have described previously in the Scandinavian Journal of Therapy in 1918. (Nordisk tidskrift for Terapi.)

This instrument consists of the following parts:

1) A dental plate for fixation against the teeth. (Fig. 1.)

2) A pivot-joint piece, the pivots of which fit into the notches of the dental plate and admit of movements round a vertical axis. (Fig. 1.)

3) A hook-like lock-arrangement. (Fig. 1.)

4) The cylindrical inner arm of the tongs with the radium plate. (Fig. 2.)

5) The outer arm of the tongs with a pelotte against the outside of the cheek. (Fig. 2.)

This instrument allows the plate holding the radium preparations to move in all directions. This plate may, thus, be moved and shifted

1) forwards and backwards, 2) outwards and inwards, 3) upwards and downwards, and, finally, 4) rotation may take place.

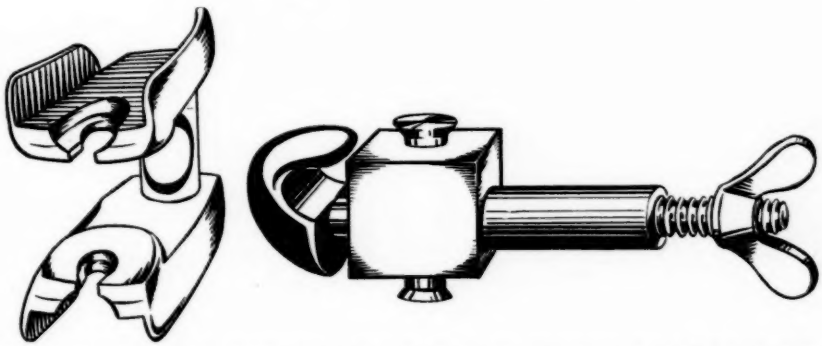


Fig. 1. The dental plate for fixation against the teeth; the pivot-joint piece with the hook-like lock-arrangement. The pivots fit into the notches of the dental plate. The hook is movable round its longitudinal axis towards the rectangular piece. The hook locks the cylindrical inner arm of the tongs, when it is pressed against the rectangular piece, by tightening the winged-screw.

The instrument may be used on the right as well as on the left side. It is very easy to take to pieces and to clean. All its lines are simple and straight. By dividing the movements round three axes and in one plane I have managed to avoid the ball-joint, which is a very unsuitable joint, because it is difficult to take to pieces and hard to clean and therefore easily becomes rusty, and also because a locking of the ball-joint will never be quite secure.

The instrument is made by the Surgical Instrument Company of Stockholm. (Kirurgiska instrumentfabriksaktiebolaget, Stockholm.)

The application is done in the following manner:

The treatment is given most advantageously on an empty stomach or, at the very earliest, 4—5 hours after partaking of food. Before

the treatment is commenced, the patients receives 1/2 milligram of atropin subcutaneously to prevent a hindering salivation and phlegm formation. In some few cases I have given nervous patients 1 c. gr. of pantopon subcutaneously. The extension of the tonsillar surface is definitely ascertained after the pharynx has been cocainized by painting with a 20 % solution of cocain in grown-up people and a 20 % solution of novocain in children. A few drops of adrenalin are added to the solutions. The radium tubes are fixed on the plate of the inner arm by means of Kerrs plastic dental mass. The small plate constitutes then a protective filter back towards the uvula and the opposite side of the pharynx. The dental plate is fixed by means of plastic mass against the molars of the upper and lower jaws, which is done by one single manipulation. The upper

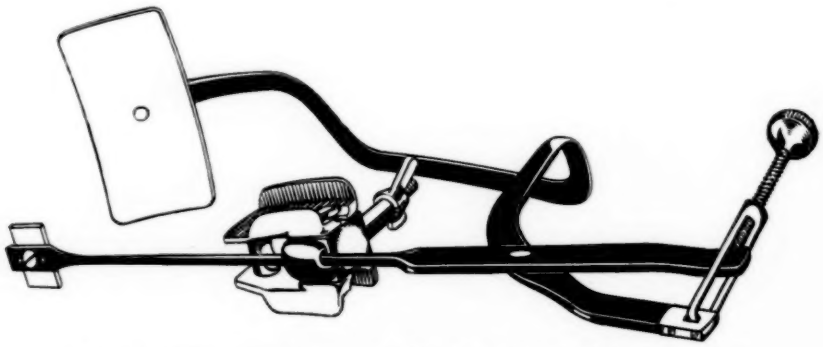


Fig. 2. The application arrangement with all its pieces in their proper places.

and lower jaws are fixed against each other by winding a bandage a couple of turns round the top of the head and the chin, or still better by the following arrangement:

On the chin is fitted a metal chin-plate, the inner surface of which is coated with plastic mass, which fashions itself exactly after the shape of the chin. A round metal ring, having a bed of cotton-wool to make the pressure less unpleasant, is placed round the top of the head. The chin plate and the head ring are fixed to each other by means of a strap running between four hooks.

I can now easily get a good view of the pharynx through the mouth, which is thus fixed in a comfortable, half-open position. The inner arm of the tongs holding radium is now introduced into the mouth, is fitted into the hook-shaped lock-arrangement, and is then carried backwards until it reaches the tonsil. The final exact

adjustment is easily done under ocular control by rotatory movements and upward and downward movements round the two horizontal axes, from before backwards and from side to side. As soon as the radium preparation well covers the surface of the tonsil, the winged-screw on the outside is then tightened and all the joints are locked except the pivot joint. The outer arm of the tongs

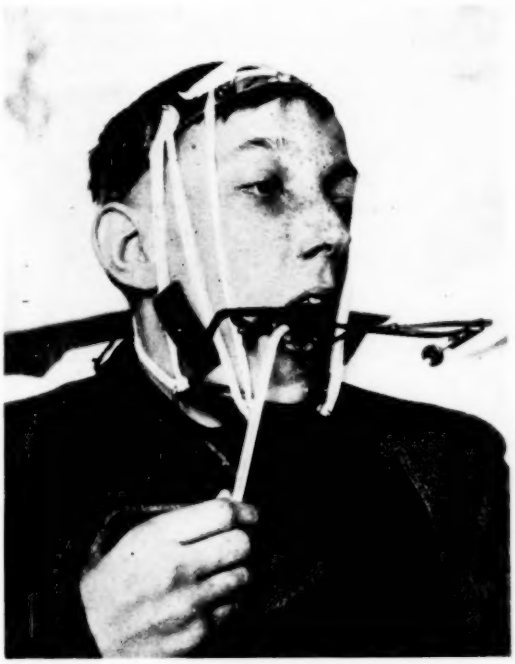


Fig. 3. A patient during treatment.

is then fitted on, and finally also the pivot joint is locked by pressing the two arms together.

Fig. 3 shows the patient during treatment.

II. For the application of radium in the nasopharynx ALBANUS, WICHMAN, LEDERMANN and KUZNITSKY, FREUDENTHAL, FINZY, DAWSON, TURNER and EDLING have described different methods. The most simple of these methods is the manual application where the patient himself or a nurse presses the radium up against the nasopharynx, the radium being secured to a holder. Others recommend placing the

radium in a soft catheter and introducing this into the nasopharyngeal cavity. Most authors introduce the radium tube into the nasopharynx and fix it there by means of a hard tampon. The fixation is, however, not quite reliable, and it is difficult to control whether the radium preparations are lying in their proper place. Thus, LEDERMANN and KUZNITSKY, who employ this method, have described some cases of severe injuries by radium with perforation of the soft palate. I am still using the method described by me in Nordisk Tidskrift for Terapi in 1918.

Fig. 4. Applicator for treating the lymphoid tissue of the nasopharynx. Thick lead protection of the soft palate. The radium tubes lying across the nasopharynx. With the aid of the two threads fixed to the front part of the applicator this is drawn up into the nasopharynx and is removed after the treatment by pulling the hind thread.

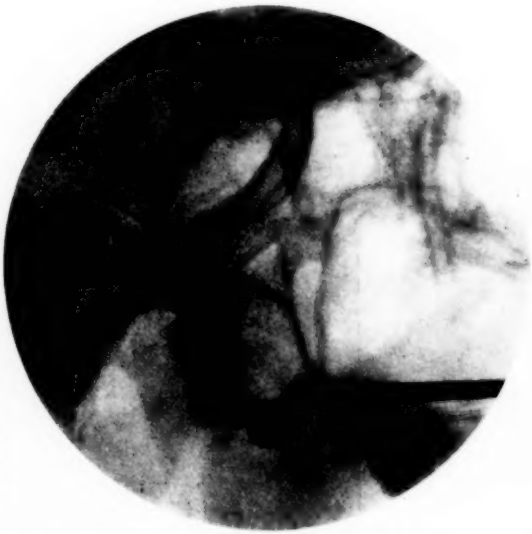


Fig. 5. Roentgen picture showing the position of the radium tubes during treatment of the lympoid tissue in the uppermost part of the nasopharynx. The tubes are placed vertically on either side of the nasal septum.

After a careful examination of the nasopharynx I fashion an applicator out of Kerr's plastic mass, the applicator coinciding as far as possible with the conditions found at the examination. I place a thick protective filter on the concave surface which is meant to lie against the palate. The radium tubes are then placed crosswise on the convex surface lying against the pharyngeal tonsil. Two silk threads are fixed anteriorly and one posteriorly. (Fig. 4.)

A thin catheter is introduced through either nostril into the nasopharynx and then down into the oropharynx where the end of the catheter is grasped by means of a forceps and pulled out through the mouth. The two silk threads which are fixed to the front part of the applicator, are now tied to the ends of the catheters, after which the applicator is easily drawn through the buccal cavity and the oropharynx up into the nasopharynx, in the same manner as a posterior tampon is applied. It is then secured in its proper position by the two threads being tied together across the anterior border of the nasal septum, which is protected against the pressure by means of a rubber tube lying across the septum. By this applicatory device I manage to get an absolutely reliable fixation and a very good protection of the soft palate. (Fig. 5.) Fig. 4 and 5 show the application arrangements.

III. In distance treatment a layer of wood, or plastic mass, is usually placed between the skin and the radium preparations. In using this application arrangement it is, however, difficult to protect the patient sufficiently.

In Radiumhemmet we have therefore an application arrangement adapted for distance-treatment and devised by Dr. LYSHOLM.

The radium preparations are placed in a lead cylinder with 5 c. m. thick walls. The extent of the radiation field is regulated by means of a diaphragm in such a way that the radiation only falls on the area to be treated, whereas the rest of the body of the patient is well protected. The contrivance holding the radium is fixed to a stand and is movable in relation to this stand by means of a ball-joint. The adjustment may be controlled at a glance by means of a pointer which moves in the direction of the central ray, and which is adjusted towards the centrum of the treatment area.

The technic of dosage.

I. The radium preparations that I have used in the treatment, have been as follows:

1) Two tubes consisting of an inner tube of gold having a metal thickness of 0.35 mm. and an outer tube of platinum with a metal thickness of 0.3 mm. The outer tube measures 15 mm. in length and has a diameter of 2.8 mm. The inner tube of either preparation contains 23 milligrams radium element, the amount being ascertained by measuring the γ-radiation after it has passed through the tube wall.

2) Two tubes which also consist of an inner tube of gold and an outer tube of platinum with a metal thickness of 0.4 and 0.3 mm. resp. The outer tube measures 20 mm. in length and has a diameter of 3.25 mm. Each of these tubes contains 25 mgr. radium element.

3) Two tubes likewise consisting of an inner tube of gold and an outer tube of platinum, the walls being 0.35 and 0.30 mm. resp. The tubes have a length of 25 mm. and a diameter of 2.8 mm. Each tube contains 23.5 mgr. radium element.

The filtration capacity of the tube wall corresponds to a filtration capacity of one millimeter of lead, and through this practically the whole of the β-radiation and 6 % of the γ-radiation is filtered off.

The dosages employed by me in the radium-treatment are as follows:

II. 1) The majority of the patients with large, soft tonsils, or 123, have received a treatment in which the dosage varies from 240 to 288 mgr.-hours. The patients have had, then, 96 mgr.-hours radium element for $2^1/_2$ and 3 hours resp. For tonsils with a very large surface I have used all six tubes, e. g. 143 mgr.-hours Ra.El. for 2—$2^1/_2$ hours. The dosage has in this case amounted to 288—357.5 mgr.-hours Ra.El. resp.

I shall describe later on the reaction obtained by this dosage. The course of reaction is hardly altered at all by the above-mentioned variations of the dosage.

The tubes have been fixed to the radium-carrying plate by means of Kerr's plastic mass, and a 1 mm. thick layer of this mass has been applied between the tubes and the tonsilar surface.

2) On five patients who have had very large and fibrous tonsils, I have employed a different technic with a dosage exceeding 288 mgr.-hours Ra.El. and amounting to 800—1000 mgr.-hours Ra. El. In employing this dosage I have, however, filtered the tubes through an additional lead filter of 1 mm. and placed a 3 mm. thick layer of plastic mass between the filter and the surface of the tonsil. By this technic I have increased the distance considerably and have filtered off the most of the secondary soft β-rays which reached the surface in large numbers at the previous dosage.

3) Some few cases (10) have been treated by the same technic as the majority, but the dosage has been somewhat lessened, down to 150 mgr.-hours. This dosage has been used for small tonsils, or when, because of outward circumstances, I have had to treat both sides within one or two days of each other.

As to the results of the treatment by the different dosages I shall enter more fully into these in connection with the discussion on the size of the dosage.

4) Five patients have got distance-treatment. I have then used about 300 mgr. of radium for 10—11 hours, the whole dosage amounting to 3000—3300 mgr.-hours. The area of the radiation field comprises a rectangle, the lower limit of which lies about 1 cm. below the angle of the jaw and its upper limit 1 cm. above the zygomatic arch. From before backwards the radiation field measures about 4 cm.

5) The dosage employed by me in the treatment of the pharyngeal tonsil has varied very much because of the considerable variations in the size of the tonsil and the different conditions of application.

In the majority of cases I have applied 2 tubes for a time of 3 hours, which is equivalent to a dosage of 138 mgr.-hours. In some cases, I have used the ordinary tonsillar dosage of 288 mgr.-hours. Finally I have treated two cases with a larger dosage up to 500 mgr.-hours, but the tubes were then provided with an additional lead filter of 1 mm.

III. Each tonsil is treated only once; the other tonsil 4—5 weeks after the first one.

The course of reaction during and after the treatment.

I. After the preparation has been applied against the tonsil and the fixation is completed, the patient is relatively not very much inconvenienced by the treatment.

The pressure of the tongs is, in itself, only slightly inconveniencing. The secretion of saliva is inhibited by the injection of atropin, but should it now and again become excessive, the saliva is sucked out by means of the ordinary suction-pump employed by the dentists.

The patient is able to sit reading during the treatment. He is a little tired afterwards and feels a lump in the throat, but several patients have gone straight to their work after the treatment.

II. During the day of the treatment and on the following day the patients feel a certain tenderness in the throat, which feeling then disappears. Then, as a rule, no subjective troubles from the throat appear until the seventh or eighth day, when some burning together with tickling and pricking sensations set in. On the ninth day the troubles increase so that the patients have some difficulty in swallowing. They state that the troubles are identical to light anginal troubles. On the other hand, they do not have any direct feeling of illness at all and the temperature is quite normal.

They need not stay away from their business or from school. These angina troubles then continue for 2—3 days until the 12th or 13th day after the treatment, when a distinct relief sets in. The abatement of the troubles is very rapid, takes only about 2 days. The duration of the subjective reaction lies, then, between the 7th and the 15th day after the treatment and, thus, comprises about one week. This subjective reaction can, however, be very much lighter, particularly in children, and some have no trouble at all in swallowing, but only a slight burning sensation.

Quite subjectively, the patient has himself been able to notice a diminution of the tonsils, which occurs simultaneously with the reaction, in as much as they have felt how the air current in breathing has passed through more easily on the side of the pharynx where the treatment has been given.

III. 1) With regard to the objective reaction, careful observations have been made from day to day. At the end of the treatment the entire pharynx appears somewhat reddened and the treated tonsil is distinctly swollen. In some cases with very loose tonsils the treated tonsil has, however, been somewhat reduced in size already at the end of the treatment. The swelling has subsided completely on the first day after the treatment and there remains only an insignificant redness. Then the tonsil remains almost unchanged until the 9th day, thus, a couple (2—3) of days after the subjective troubles have started. From the 9th to about the 11th—13th day a membranous coating begins to appear on the tonsil where the radium tubes have been lying close against its surface. This membrane formation starts in the form of small round spots which gradually spread out, the whole surface being coated with this membrane after 3—4 days. The membrane becomes visible, on the 9th day at the earliest and at the latest on the 11th day. After having lasted for about 6 days, the membrane begins to get thinner at its edges as well as all over its surface. One sees red surfaces shining through the membrane in its entire width. The membrane disappears completely within 2—3 days and on the 20th—23rd day there is no sign of the membrane to be seen at all.

Thus, the objective reaction with membrane formation sets in a few days after the commencement of the subjective troubles and remains for about 7 or 8 days after the abatement of subjective symptoms.

The membrane is easily removed by wiping the surface with some cotton-wool. Then a grayish-red surface which does not bleed, appears beneath the membrane.

The surface of the tonsil becomes covered with epithelium at the

same time as the membrane becomes mellow and disappears. At the edge of the membrane one notices a faintly red and slightly edematous zone of the mucous membrane, apparently a small reaction zone.

2) The course of reaction at a dosage of about 1000 mgr.-hours, filtered through an additional lead filter of 1 mm., is essentially the same as those previously described, though the membrane remains for a longer time, the membrane being dissolved completely only after 4 weeks or thereabouts.

3) At a dosage of between 150—200 mgr.-hours the subsequent radium reaction becomes very slight, and one can only find a faint redness which appears after about 14 days. No distinct membrane can be seen at this dosage.

4) In the distance-treatment I have not been able to find any reaction, either on the skin or in the throat. The tonsils have begun to decrease in size during the 3rd week, but this diminution in size has not reached any high degree.

On examining the cases two years after the treatment not the slightest change can be seen on the skin.

IV. After the treatment of the pharyngeal tonsil the patient has no sensations at all of the radium reaction in progress. The objective changes are also very insignificant. In occasional cases one may find a thin yellowish-white membrane just where the radium tubes have been lying most closely against the surface. The reason of this light reaction is apparently to be found in the fact that the distance, at which the treatment is taking place, is greater than in the treatment of the palatine tonsils, as one is not able to effect any pressure against the tonsillar surface in this case. Already in the second week one is able to observe the diminution in size, which then progresses for about 4—5 weeks.

Classification of the material.

I. The material that I have had at my disposal during my investigations, consists of patients who have visited the outpatients' department of Radiumhemmet for throat complaints and also of patients sent from other physicians to be treated. The number of treatments given has increased in the course of years. Thus in 1919 I treated 3 cases, in 1920 15 cases, in 1921 29 cases, in 1922 84 cases and during the first months of 1923 23. Altogether 154 patients have, thus, been treated for chronic tonsillitis or symptoms secondary to chronic tonsillitis.

I have put down the diagnosis »chronic tonsillitis» in such cases

Group	Results of treatment	Dosage > 288 mg. h. Ra. EL.		Dosage 240—288 mg./h. Ra. EL.					Dosage < 240 mg./h. Ra. EL.			
		1921	1922	1919	1920	1921	1922	1923	1919	1920	1921	1922
I	Compl. treated. Symptomless	1		1	4	6	24	10				2
	Partially treated. Symptomless		1		2	5	6	1				
	Partially treated. Part. symptomless				1	1	3					
	Compl. treated. Symptoms left					2						1
	Partially treated. Symptoms left						2					
	Treatment abandoned											
	Roentgen treated											
II	Compl. treated. Symptomless		1				1	4				
III	Compl. treated. Symptomless			1	1							
IV	Compl. treated. Symptomless						1					
	Compl. treated. Symptoms left						2					
V	Compl. treated. Symptomless					2	1	2	1			1
	Partially treated. Symptomless						1					
	Compl. treated. Symptomless locally. Second. symptoms left									1		
VI	Compl. treated. Symptomless	1			1		4	1				
	Partially treated. Symptomless				1							
VII	Treatment abandoned											
VIII	Compl. treated. Symptomless						2					1
	Partially treated. Symptomless					1	1					1
IX	Partially treated. Symptomless				1	1						
X	Compl. treated. Symptomless				1	2	7				1	
	Partially treated. Symptomless				1	1	1					
	Compl. treated. Symptomless locally. Second. symptoms left						2			1		
	Partially treated. Symptomless locally. Second. symptoms left							1				
XI	Compl. treated. Symptomless						2	1				
	Partially treated. Symptomless						1					
XII	Treatment abandoned											
XIII	Compl. treated. Symptomless	1						1				
	Partially treated. Symptomless					1						
XIV	Partially treated. Symptomless					1	1					
	Partially treated. Symptoms left						1					
XV	Partially treated. Symptomless						1					
		3	2	2	13	23	64	21	1	2	1	6
		5		123					10			

Fig. 6. Tabula, showing the results of the treatment, the patients being

Distance treatment		Roentgen treatment		Treatment abandoned	Compl. treated. Symptomless	Part. treated. Symptomless	Part. treated. Part. symptomless.	Compl. treated. Symptomless locally Second. symptoms left	Part. treated. Symptomless locally Second. symptoms left	Compl. treated, Symptoms left.	Part. treated, Symptoms left.	Roentgen treated	Treatment abandoned
1921	1922	1922	1923	1922	Total	Total	Total	Total	Total	Total	Total	Total	Total
1	2				51	15							
							5						
										3			
											2		
				4									4
		2	2									4	
					6								
					2								
					1								
										2			
					7								
						1							
								1					
	1				7								
						2							
				2									2
					3								
						3							
						2							
1					12								
						3							
								3					
									1				
					3								
						1							
				1									1
					2								
						1							
						2							
											1		
						1							
2	3	2	2	7	94	31	5	4	1	5	3	4	7
5		4											

classified according to the size of the dosage and to the group of treatment.

Group	1919				1920							19						
	I	III	V	Total	I	III	V	VI	IX	X	Total	I	II	V	VI	VIII	IX	X
Compl. treated. Symptomless	1	1	1	3	4	1		1		1	7	8		2	1			4
Partially treated. Symptomless					2			1	1	1	5	5				1	1	1
Partially treated. Part. symptomless					1						1	1						
Compl. treated																		
Symptomless locally							1			1	2							
Second. symp. left																		
Partially treated																		
Symptomless locally																		
Second. symp. left																		
Compl. treated. Symptoms left												2						
Partially treated. Symptoms left																		
Roentgen treated																		
Treatment abandoned																		
				3							15							

Fig. 7. Tabula, showing the results of the treatment, the

where the anamnesis of the patient has exhibited numerous recurring attacks of anginas or throat abscesses and where, on examination, the tonsils have been found distinctly enlarged with rough surface and numerous crypts from which exudates could be pressed out.

With regard to the symptoms which, together with the troubles usually associated with the chronic tonsillitis, have been predominant in the clinical history, I have divided the material into 15 different groups.

Group I: *Cases of frequently recurring angina, tonsillar and peritonsillar abscesses.*

Group II: *Cases of hypertrophic pharyngeal tonsil with concomitant local symptoms.*

Group III: *Cases of catarrhs in the Eustachian tube.*

Group IV: *Cases of diffuse hypertrophy of the adenoid tissue of Waldeyer's ring, and symptoms of diffuse pharyngeal catarrh.*

Group V: *Cases of recurring angina in combination with neuralgic pains.*

Group VI: *Cases of recurring angina and acute rheumatic arthritis, developed in connection with angina.*

Group VII: *Cases of chronic arthritis deformans without distinctive angina in the anamnesis.*

Group VIII: *Cases of recurring angina and vitium organicum cordis.*

Group IX: *Cases of angina and subchronic lymphadenitis.*

Group X: *Cases of recurring angina and tuberculous lymphomata of the neck.*

2 1			1 9 2 2												1 9 2 3								
XIII	XIV	Total	I	II	IV	V	VI	VII	VIII	X	XI	XII	XIV	XV	Total	I	II	V	VI	X	XI	XIII	Total	
1		16	28	2	1	2	4		3	7	2				49	10	4	2	1		1	1	19	94
1	1	10	7			1	1		2	1	1		1	1	15	1							1	31
		1	3												3									5
										2					2									4
																				1			1	1
		2	1		2										3									5
			2										1		3									3
			2												2	2							2	4
			4					2				1			7									7
		29													84								23	154

patients being classified according to the year of treatment.

Group XI: Cases of recurring angina in combination with symptoms from the kidneys.

Group XII: Cases of recurring angina and cystitic troubles.

Group XIII: Cases of recurring angina, in which the patient has suffered from hemorrhagic diathesis at the same time.

Group XIV: Cases of various complaints remaining after tonsilectomy.

Group XV: Cases of unilateral tonsillar hypertrophy, causing obstinate vomitings.

II. In the synoptical table, Fig. 6 p. 22—23 I have grouped the results together under the following headings:

1) Completely treated. Symptomless. 2) Partially treated. Symptomless. 3) Partially treated. Partially symptomless. 4) Completely treated. Symptomless locally. Secondary symptoms still left. 5) Partially treated. Symptomless locally. Secondary symptoms still left. 6) Completely treated. Symptoms left. 7) Partially treated. Symptoms left. 8) Roentgen treated. 9) Treatment abandoned.

By *completely treated* I mean those patients who have had treatment of both of the tonsils and of the pharyngeal tonsil, when the latter has exhibited any symptoms. By *partially treated* I understand those patients who have had treatment of one tonsil only.

By *partially symptomless* I mean those patients who, having had only one tonsil treated, have become free from symptoms on that side, whereas now and again they have had slight attacks of angina on the untreated side.

The patients are classified according to the dosage they have received as well as to the year of treatment.

In the synoptical table fig. 7, pag. 24—25, I have grouped together the results of treatment obtained in the various groups during the different years.

The results of treatment.

Group I: Cases of frequently occurring angina, tonsillar and peritonsillar abscesses.

1. The regularly repeated anamnesis of this group is about as follows:

Ever since his childhood the patient has frequently been troubled with sore throat. Every spring and autumn the patient has had almost regularly a general feeling of illness with head-ache, feverishness, soreness and pains in the throat, difficulty in swallowing and tenderness on pressure behind the angles of the jaws. On inspection the mucous membrane appeared reddened and the palatine tonsils swollen and often coated with a grayish-white membrane. The patient has been feeling really ill and has been obliged to go to bed. On the temperature being taken it has been found to be as high as 39°—40° C.

After 2—6 days the troubles have become less and after another couple of days the patient has again felt quite well. To children in the school age these repeated attacks of angina mean an absence from school for 6—10 days every time. To older people these attacks are attended with a considerably lowered working power for the same length of time.

The angina attacks have sometimes a rather lengthy course, especially in young children, the temperature returning but slowly back to the normal state, and the general condition seems then to be much more affected. It is definitely stated in some cases that the tonsils have not returned to their normal size after an attack of angina, but have increased somewhat in size every time. It is a remarkable fact that the attacks of angina are generally concentrated to the dark seasons with changeable weather, whereas the patients are usually rid of their throat troubles during the bright summer months. The anginas become often more troublesome with increasing age, and in my material the severe angina cases and the throat abscesses appear about the age of fifteen, to be about evenly distributed to the age of thirty. Those patients who have once had a tonsillar or peritonsillar abscess get as a rule always a relapse of this complaint. Thus, one of the patients, a male aged 29, has suffered

for the last twelve years before the treatment, from abscesses recurring regularly every autumn and spring.

2. On examination of the patients belonging to this group, the tonsils appear greatly enlarged in nearly all the cases, and especially in children, in whom they often become so large as to leave only a narrow slit between the medial surfaces. More rarely do the tonsils attain such a high degree of enlargement in older persons. The surface is fissured and uneven, and with a probe one can easily get into crypts 3—4 m. m. deep. In most cases there is a deep pocket between the anterior and posterior pillars and the tonsilar surface, in which depression foul-smelling food-rests often collect in great quantities. By squeezing the tonsil, quite a number of plugs may be pressed out from the crypts. The consistence of the greatly enlarged tonsils is very soft during childhood. Generally it gets gradually more firm with increasing age. In patients who have suffered repeatedly from abscesses in the throat one finds often a deformity of the tonsil, and the faucial pillars have deep fibrous scars on their surfaces. The tonsilar tissue may be completely fissured by deep-going, cicatricial connective tissue streaks.

The hypertrophy is very often not limited to the palatine tonsils. In many cases one sees a distinct thickening especially of the posterior pillar, and behind this, plenty of adenoid tissue may be observed. The contour of the pillars is sometimes irregular (see for instance fig. 35 Tabula IX) owing to infiltrations of adenoid tissue.

3. The vast majority, or 84 cases out of my material, belong to this group. Thirteen of these have at the same time had considerably enlarged pharyngeal tonsils with the characteristic symptoms — mouth breathing, snoring, troubled sleep etc. This hypertrophy of the pharyngeal tonsil is often found in patients up to the first half of their second decade. Then it begins to occur more sparingly. In this group only one patient over fifteen years has an enlarged pharyngeal tonsil.

Seventy-one (fig. 6. I) out of the eighty-four patients treated in this group have derived the desired benefit by the treatment in so far as the tonsils have been reduced to their normal size and have been lying entirely within the fauces. The surface of the tonsil which before the treatment was very much fissured by numerous crypts from which a great many plugs could be pressed out, has become smoother and the crypts have disappeared to a large extent.

All the patients who have suffered from tonsillar or peritonsillar abscesses, have not been troubled with anything of that sort since the treatment was begun. In some cases, in which the patient has been inconvenienced by foodrests collecting in the pockets between

the tonsils and the pillars and thereby producing foul breath, this symptom has disappeared completely, and one is no longer able to press out such large amounts of plugs.

Five patients still have their symptoms but two of them are distinctly improved. I will now give a more detailed account of these cases.

Case 1 relates to a girl, aged 8, who was treated with the ordinary dosage, in Dec. 1921. Before the treatment she used to suffer repeatedly from lengthy attacks of angina with rise of temperature up to 40° C. and with an impaired general condition after the attacks. Her general condition has improved considerably after the treatment, but she has had a relapse in the autumn 1922. This attack was, however, far lighter and shorter than the previous one and started from the left tonsil which was reduced to medium size by the treatment, whereas the right one was more reduced.

Case 2 relates to a man, aged 40, whose anamnesis exhibited numerous anginas, with a diffuse hypertrophy of the mucous membrane of the whole pharynx: In this case the treatment has had absolutely no effect as he is still troubled with angina to the same extent as before the treatment. The tonsils have not been reduced in size after the treatment.

Case 3 relates to a woman, aged 25. She was treated in June 1922 with a dosage smaller than the usual one, viz. 191 and 240 mgr.-hours. She is still troubled with abundant exudate and has had repeated attacks of angina, of which only one, however, has had the same intensity as the previous attacks.

Case 4 has reference to a woman, aged 28, who has had treatment only of the right tonsil which became reduced in size, the patient continuing, however, to be inconvenienced by exudate plugs from the tonsil. On closer examination I found a crypt which I probed to the depth of 6 m. m. and from which numerous plugs could be pressed out. A radium tube was introduced into this crypt, dosage 23 mgr.-hours, without any real decrease in the formation of plugs being noticeable.

Case 5 relates to a man, aged 29, who has only had one partial treatment, viz. of the left tonsil. He has since then had one attack of angina which was less intense than his previous one, and concentrated mostly to the right side.

Four patients have discontinued the treatment, and I have not been able to find out their present condition.

Thus, out of the 84 treated patients belonging to this group are:

Symptomless	71
Improved	2
Symptoms left	3
Roentgentreated	4
Treatment abandoned	4

4. As regards the diminution in size of the tonsils one may observe in some cases with very large and loose tonsils a certain diminution almost immediately after the treatment. In the majority of cases, however, the reduction in size starts about the time when

the subjective symptoms of the reaction have ceased, thus, about the 14th or 15th day, and then continues slowly.

When the fibrinous membrane has been dissolved, about the 23rd or 24th day after the treatment, the tonsil has, as a rule, been reduced by about one third or one half of its original size, and about one month or six weeks after the treatment, it has shrunk to a small remainder which then, as a rule, lies completely within the fauces. It is surprising to see the difference between the size of the tonsils before the commencement of the treatment and then six weeks later. In cases where the tonsils before the treatment were so large that they were altogether predominating in the isthmus and covered the posterior pillar to such an extent that only the upper 2 or 3 m. m. of its outline were visible, or in cases where the tonsils at vomiting movements welled out from the fauces as large egg-shaped forms, or where the surface before had been deeply reddened and fissured, and had contained deep crypts from which numerous plugs could be pressed out, the tonsils now appear as bean- or almond-sized structures, lying within the fauces. One can see how the whole of the posterior pillar which often was irregular and thickened by adenoid tissue, is now smooth and regular. The surface of the tonsils presents a pale, smooth appearance and the crypts are far less numerous. In a great number of the cases one is no longer able to press out any plugs from the tonsils.

In the figures 8—31, pag. 30—31, I have shown by diagrams the reduction in the size of the tonsils of 23 treated patients of various ages, belonging to the various groups.

Under each figure I have stated the days of treatment, and the day, on which the figure was sketched, together with the dosage.

Figs. 35—42 on Tabulae IX and X show the condition of the tonsils at various times after the treatment.

5. The results have been permanent as far as the disappearance of the anginal attacks is concerned. Those patients who have had a negative result of the treatment, have got their attacks of angina at the time as they used to get them, without first having been symptomless.

As regards the diminution in size I have found that it has been permanent during the period of observation in nearly all the cases. In some patients over 30 years of age a slowly progressing diminution has taken place and has finally resulted in a complete disappearance of the tonsil. In children, on the other hand, I have noticed in some cases that the tonsil, after having shrivelled to normal size and being located in the fauces, has increased up to average size after a year or more. This has, however, not been attend-

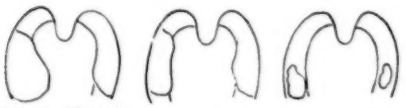
17. 2. 20. 15. 12. 19.
13. 12. 19. 2. 1. 20. 15. 1. 20.
R. tons. 101 mgr × 2 h.
L. tons. 96 mgr × 2 h.
Group 5. Aged 38.

Fig. 8.

30. 1. 20. 17. 2. 20. 22. 3. 23.

Same patient as above.

Fig. 9.



22. 8. 21. 17. 10. 21.
19. 8. 21. 14. 9. 21. 3. 2. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 1,5 h.
Group 10. Aged 14.

Fig. 10.

19. 1. 21. 3. 1. 21.
19. 1. 21. 19. 10. 21. 2. 10. 22.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 2,5 h.
Group 5. Aged 34.

Fig. 11.

12. 12. 21. 11. 2. 22.
12. 12. 21. 9. 2. 22. 2. 5. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 119 mgr. × 3 h.
Group 1. Aged 42.

Fig. 12.

13. 10. 22. 29. 1. 23.
12. 10. 22. 8. 1. 23. 9. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group 6. Aged 9.

Fig. 13.

29. 8. 22. 13. 10. 22.
29. 8. 22. 28. 9. 22. 15. 3. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group 1. Aged 11.

Fig. 14.

7. 12. 22. 26. 10. 22.
26. 10. 22. 30. 11. 22. 4. 1. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group 1. Aged 11.

Fig. 15.

29. 11. 22. 3. 1. 23.
29. 11. 22. 3. 1. 23. 4. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group 10. Aged 11.

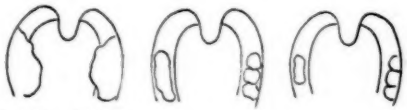
Fig. 16.

17. 10. 22. 13. 11. 22.
17. 10. 22. 11. 1. 23. 22. 3. 23.
R. tons. 96 mgr. × 3 h.
L. tonr. 96 mgr. × 3 h.
Group 1. Aged 15.

Fig. 17.

9. 3. 22. 15. 4. 22.
14. 1. 22. 8. 3. 22. 11. 5. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 17.

Fig. 18.



1. 4. 22. 21. 1. 22.
26. 1. 22. 1. 4. 22. 22. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group VI. Aged 17.

Fig. 19.

Figs. 8—31 show the diminution in the size of the tonsils in 23 patients of various ages and the sketch was made

2. 11. 22.
2. 11. 22. 28. 12. 22. 18. 1. 23.
R. tons. — — — — —
L. tons. 96 mgr. × 3 h.
Group I. Aged 17.

Fig. 20.

29. 9. 22. 19. 2. 23.
29. 9. 22. 21. 2. 23. 2. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 21.

Fig. 21.

30. 11. 22. 5. 1. 23.
30. 11. 22. 5. 1. 23. 13. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 143 mgr. × 2 h.
Group I. Aged 22.

Fig. 22.

27. 8. 22. 2. 2. 23.
26. 8. 22. 8. 3. 23. 2. 5. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 23.

Fig. 23.

20. 10. 22. 11. 3. 22.
10. 3. 22. 21. 10. 22. 30. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 2,5 h.
Group I. Aged 27.

Fig. 24.

23. 3. 22. 22. 6. 22.
23. 3. 22. 16. 6. 22. 7. 8. 22.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group IV. Aged 39.

Fig. 25.

2. 3. 23. 31. 3. 23.
1. 3. 23. 30. 3. 23. 19. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 9.

Fig. 26.

8. 2. 23. 14. 3. 23.
8. 2. 23. 14. 3. 23. 30. 4. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 11.

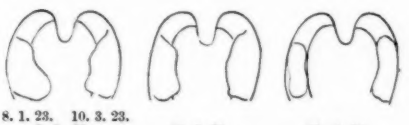
Fig. 27.

6. 3. 23. 10. 4. 23.
5. 3. 23. 23. 3. 23. 2. 5. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 24.

Fig. 28.

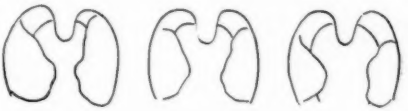
23. 4. 23. 16. 3. 23.
16. 3. 23. 5. 4. 23. 3. 5. 23.
R. tons. 96 mgr. × 3 h.
L. tons. 96 mgr. × 3 h.
Group I. Aged 29.

Fig. 29.



8. 1. 23. 10. 3. 23.
8. 1. 23. 15. 3. 23. 29. 3. 23.
R. tons. 143 mgr. × 2 h.
L. tons. 120 mgr. × 3 h.
Group I. Aged 43.

Fig. 30.



19. 10. 22. 7. 12. 22. 18. 1. 23.
Roentgen treated on both sides with 3 series.

Fig. 31.

belonging to different groups. The day of the treatment and the dosage as well as the day when are also recorded.

ed by any fresh attacks of angina, but it only shows that a vigorous regeneration of the tonsilar tissue is taking place. However, I have not had any opportunity to examine these tonsils anatomically.

Group II. Cases of hypertrophic pharyngeal tonsils with concomitant local symptoms.

In this group I have brought together 6 patients in whom the symptoms from the palatine tonsils have not been particularly marked and who have not had the common angina troubles in their anamnesis. They have come for treatment because of difficulties in breathing through the nose, obstinate nasal catarrhs, troubled sleep and snoring.

On examination the pharyngeal tonsil has been found to be greatly enlarged. The palatine tonsils have been moderately enlarged in all cases, but as they have not caused any clinical symptoms I have not treated them.

All these patients have been relieved of their subjective complaints. Already after one week or so the patients state that they are able to get their breath more easily through the nose, and after about 3—4 weeks their appearance with the open mouth and marked mouth-breathing has become more normal. At the same time there is an obvious improvment of the previously very troublesome nasal catarrh.

The parents of the patients state that the patients gradually cease sleeping with their mouths open and that the snoring is diminished. The restlessness, which used to trouble their sleep, generally also disappears at the same time.

At the objective examination one notices a reduction in the size of the pharyngeal tonsil, already from the 8th to the 10th day. In the cases treated it has been reduced to the size of a small hazelnut or bean. The surface appears even and smooth, having the colour of a normal mucous membrane.

Besides these six patients I have treated the pharyngeal tonsil of another 28 patients in the other groups, in connection with the treatment of their tonsils, viz. of 13, 2, 2, 3, 6, 1 and 1 patients in the groups I, III, VI, VIII, X, XI and XIII respectively. Altogether the pharyngeal tonsil has, thus, been treated in 34 cases.

Group III. Cases of catarrh of the Eustachian tube.

This group comprises only two patients, who were sent by an ear-specialist to be treated for a catarrh of the Eustachian tube

which would not yield to the ordinary otological treatment. In the first case there was abundant adenoid tissue in the nasopharynx and in the walls of the Eustachian tube, together with enlarged palatine tonsils. In the second case no well-defined pharyngeal tonsil could be seen but the walls of the tube showed a distinctly thickened mucous membrane. There were troublesome anginas in the anamnesis of both cases. Both of them have become free from symptoms with regard to the anginas as well as to the defective hearing.

Group IV. Cases of diffuse hypertrophy of the adenoid tissue of Waldeyer's ring, and symptoms of diffuse pharyngeal catarrh.

Three patients have been included in this group, all of them having had marked throat troubles without having suffered from any typical attacks of angina. They complain mostly of dryness in the throat together with abundant phlegm, soreness, a permanent irritation to cough and a feeling of a lump in the throat. All three of them have been treated locally for some time by throat-specialists with paintings of the throat, but the improvement has only been temporary.

On examination they exhibited a diffuse redness of the entire pharyngeal mucous membrane and a moderate enlargement of the tonsils.

Only one of these patients has got rid of his troubles. He feels clearer and cleaner in his throat, has less phlegm than before and his voice has also become clearer, whereas the other two are still harassed by the same troubles as before.

The dryness and the secretion of phlegm has not increased after the treatment.

Group V. Cases of recurring angina in combination with neuralgic pains.

This group comprises 9 patients. The anamneses are about the same as in group I, with frequent attacks of angina and tonsillar abscesses, but neuralgic pains occur in these cases as a very characteristic additional symptom. These pains appear in conjunction with the angina but remain for a considerably longer period than the angina. The pains are piercing and radiating in character and appear mostly in the form of neuralgia of the neck and shoulders. Slight swellings of the joints, especially of the finger joints, occur also in some cases, without there having been any real acute rheumatic fever.

The tonsils of these patients have not attained any very great size. Only one of them has exhibited really large tonsils.

The treatment has been successful in all cases as far as the reduction of the size of the tonsils and the disappearing of the angina are concerned. Only in one case have the troublesome pains remained.

Group VI. Cases of recurring angina and acute rheumatic arthritis, developed in connection with the angina.

In this group I have included 9 patients, in the anamneses of whom there are one or more acute attacks of rheumatic arthritis. The first attack of arthritis has developed in connection with an angina, and the patients generally get a touch of their joint-complaint at the numerous relapses of angina, having piercing pains in the joints which become sligthly swollen.

Most of the patients belonging to this group are simultaneously suffering from a vitium, developed during the first acute arthritis.

In the majority of these cases the tonsils have not been particularly enlarged but have only attained an average size. The consistence has been rather firm.

With regard to the results obtained, all the patients treated have been relieved of their anginas and the size of the tonsils has been considerably reduced. No one has had any relapse of his rheumatic arthritis after the termination of the treatment, and the dull, aching pains which were usually felt in the joints have completely disappeared, as well as the swelling of the joints.

Group VII. Cases of chronic arthritis deformans without any distinctive angina in the anamneses.

This group comprises 2 cases both of which having suffered from a progressive arthritis deformans during many years. The disease was in both cases in a very advanced stage with extensive disturbances and ankyloses of the limb joints. Now and again there were acute exacerbations with swelling of certain joints, intense pains and a rise of temperature, without there being any marked symptoms from the tonsils on these occasions.

In neither of these cases were the tonsils enlarged, but were lying as small remains within the fauces. The surface was not fissured.

No effect whatever on the disease of the patients could be observed after the treatment, and the tonsils were unchanged.

The results of these attempts being, thus, not at all inviting to a continued treatment, I have not proceeded with it but have placed them under the category »treatment abandoned». In my opinion

there is no indication at hand as to whether the tonsils ought to be treated in such cases. In cases with such high degrees of deformities and with such numerous foci of infection in the organism as the inflamed joints, it is hardly likely that a favourable result will be arrived at by a local treatment of the tonsils.

Group VIII. Cases of recurring angina and vitium organicum cordis.

In this group I have included 6 patients, all being in their first or second decade, suffering from organic heartdisease which has developed in connection with a sustained angina. All these patients have suffered from frequently repeated attacks of angina. A clearly marked progression of the heart symptoms has taken place in one of them during one of these attacks. Their general condition was relatively bad at the commencement of treatment, and some few of them have been sent by a throat-specialist to be treated, as he considered the usual tonsillectomy to be too severe an interference.

Case I was a girl, aged 6, who got a serious endo- and myocarditis following upon an attack of angina. After that she repeatedly had severe attacks of angina with fever up to 40° C., lasting 8—14 days. The general condition of the patient became very much worse after each angina, and a noticeable insufficiency set in during the fever period.

The patient was highly cyanotic. Both palatine tonsils were greatly enlarged and exhibited numerous crypts; the pharyngeal tonsil was of average size and aggravated the breathing considerably. As the bad general condition of the patient hardly permitted a radium application, she was treated with roentgen during 1920, without nevertheless receiving a complete series. Still, she improved somewhat by the roentgen treatment and had less severe attacks of angina, but had during the winter 1921—1922 some very severe relapses with high temperature. The tonsils were still very much enlarged and had numerous crypts. In Aug. 1922 I treated the right tonsil with 192 mgr.-hours radium element. This treatment was given by means of the ordinary application, but without any local anesthetization of the pharynx. After a very moderate reaction the tonsil was reduced to about half its size. The general condition of the patient became markedly improved, and in Oct. and Dec. the left palatine tonsil and the pharyngeal tonsil were treated by the usual technic. After the commencement of the radium treatment the patient has not once had an attack of angina. When her brothers and sisters were taken ill with a severe epidemic of angina, she experienced no symptoms whatever from her throat. Now she is able to stand changes in temperature very well and she does not »catch cold». Her general condition has been improved considerably at the same time and the cyanosis is not so marked.

Case 2 relates to a girl, aged 12, with a similar anamnesis, who, after only one treatment of the left tonsil in 1921, has been completely cured from her attacks of angina. Before the treatment these attacks used to prevent her several times every term from attending school.

Case 3 was a girl, aged 13, who suffered at long periods from remittent fever, arisen in connection with angina. General condition very low. She has recovered in a marked degree after the treatment of the tonsils, palatine as well as pharyngeal. She suffers no longer from attacks of angina, and the fever periods have completely disappeared. The tonsils are diminished to normal size.

Case 4 was also a girl of 14. Tonsillotomy had been performed on one side, but the tonsillar troubles still persisted on the other side. This tonsil was relatively small and I treated it with a small dosage of 192 mgr.-hours The patient has been symptomless after this treatment.

Cases 5 and 6 relate to women, aged 29, who suffered from repeated acutizations of their endocarditis. One of them has only had a partial treatment, the other one a complete treatment of either tonsil. Both of them have hade no further relapses, and the tonsils are of normal size.

Thus, all these patients have been relieved of their recurring angina troubles. And, further, their general condition has been improved and the previous usual attacks of fever have ceased. The tonsils are reduced to normal size.

Group IX. Cases of angina and subchronic lymphadenitis.

To this group I have referred two patients who had a swelling of the lymphatic glands on one side of the neck after an attack of angina. These enlarged glands had not disappeared after the abatement of the angina. The patients visited Radiumhemmet to have the glandular swelling roentgen treated. I treated only the tonsil of that side, on which the glands were situated.

After the treatment the tonsils decreased to normal size and the glands disappeared relatively soon, leaving only some small fibrous remains. In these cases I have thus observed quite an obvious effect of the tonsillar treatment on the resorption of these subchronic lymphadenites.

Group X. Cases of recurring angina and tuberculous lymphomata of the neck.

1.) In this group I have included 19 patients, the main symptoms of whom have been some more or less diffuse tuberculous lymphomata in the neck. Some of these patients have also exhibited other symptoms of tuberculosis. Thus, one has suffered from erythema nodosum and two from recurring phlyctenosis. A fourth patient has been treated for an abdominal affection which was diagnosed as Tbc. lymphoglandulae mesenteriales, and three exhibit tuberculous changes in the lungs.

The most prominent feature in the clinical picture of this group has been the tuberculous lymphomata, but repeated attacks of angina are found in the anamnesis. Thirteen of the patients have not had any roentgen treatment on the lymphomata of the neck, whereas the remaining six have had their lymphomata roentgen treated for a longer or shorter period before the radium treatment.

In consideration of the results obtained by the treatment in this group I want to distinguish between the non-roentgen treated and the roentgen treated, as definite dissimilarities exist.

2.) The anginas occupy a very prominent position in the non-roentgen treated cases and the tonsils are in most of them considerably enlarged. Only one tonsil has been radium treated in three patients, viz. on the side where the glands were.

After the treatment all these patients have been free from their angina troubles and the enlarged tonsils have been reduced to normal size or less.

These 13 cases do not constitute a sufficiently extensive material to justify a definite conclusion as to whether and to what extent the radium treatment has any effect with regard to the tuberculous lymphomata. I have not been able to find any immediate diminution of the lymphomata in connection with the decrease in the size of the tonsils, which was the case in the preceeding group with subchronic lymphadenitis. It seems, however, as if the improvement of the general condition, which was found after the tonsils had been treated with radium, would have a favourable effect on the diminution and resorption of the glands.

The glands have slowly decreased in seven out of these 13 patients who were treated with radium only, but in none of these seven cases were the glands very large or very extensive. Roentgen treatment was given in one case in connection with radium treatment, and this combination had a very favourable effect on the glands. In three cases the tonsillar treatment has had no effect on the glands in the neck, which have developed to such an extent that it has proved necessary to resort to roentgen treatment of them, which has then had a favourable result. The glands of the two remaining cases have been reduced, but these two patients have been nursed in a sanatorium because of their lung troubles.

3.) With regard to the six patients who were roentgen treated before they started with the radium treatment it is a noticeable fact, that the frequent angina attacks became more and more rare during the course of roentgen treatment. At the objective examination I have also been able to ascertain a marked effect on the size of the tonsils. Thus, only one of these six patients has had very

large tonsils, whereas the remaining five have had average-sized tonsils. This proportion contrasts in an obvious manner with the 13 not roentgen treated patients, seven of whom had very large tonsils.

The tuberculous lymphomata in these six patients have been more widely spread, and in four cases the glands have dissolved and a fistula has been formed. Five of the cases were steadily improving already before the commencement of the radium treatment, and I dare not state definitely that the radium treatment has accelerated the process of healing. One of the radium treated patients who was treated only with 100 mgr.-hours radium element on either tonsil, has got new glands which have dissolved and which have been subjected to surgical and renewed roentgen treatment.

4.) As a final outcome of the cases treated in this group I want to emphasize the following points:

1) The patients who have been treated with radium only, have frequently had recurring attacks of angina before the treatment, but these attacks have disappeared and the tonsils decreased. The radium treatment given seems to have a favourable effect upon the tuberculous glands, when these have not attained too great a development.

2) In the patients who were treated with roentgen before being treated with radium, the attacks of angina became gradually less frequent during the roentgen treatment. No definite conclusion can be arrived at through my material as to an accelerated resorption of the tuberculous glands after the radium treatment, as most of the patients were in a progress of healing when the radium treatment was commenced.

The tonsils have decreased to normal size.

Group XI. Cases of recurring angina in connection with symptoms from the kidneys.

The four patients treated in this group have all suffered from frequent attacks of angina. An acute glomerulonephritis has developed in connection with one of these attacks. Three cases had a protracted course with a low general condition. The fourth patient suffered from a cyclic albuminuria.

The enlarged tonsils decreased considerably in all cases, a marked and speedy improvement of the disease and general condition set in along with the treatment.

Three of the patients are now completely free from their kidney symptoms. The fourth patient who only received a partial treatment, has now and again traces of albumin in his urine.

Group XII. Cases of recurring angina and cystitic troubles.

This group comprises only a 10 years old girl, suffering from an oft-recurring cystitis with coli bacilli in the urine. Anginas occur in the anamnesis, yet without it being possible to establish any definite connection between the anginas and the cystitis. The tonsils were moderately enlarged with an uneven surface. The patient received one treatment of the left tonsil with 288 mgr-hours. radium element. This treatment reduced the tonsil somewhat but had no effect whatever on the cystitis. Owing to this result the treatment was not pursued but was stopped by me.

Group XIII. Cases of recurring angina with simultaneous hemorrhagic diathesis.

The three patients belonging to this group have suffered from frequent throat troubles. All of them belong to families of bleeders and have, as a rule, abnormally excessive bleedings on lesions occuring.

The oldest of these patients, 63 years of age, had very large tonsils reaching right to the lateral border of the uvula. All her life she has been suffering from throat symptoms with frequently recurring angina, but one has not dared to perform a tonsillectomy because of the patient's disposition to bleedings. Fig. 41 tabula X shows the status praesens of the patient. Her right tonsil has been treated with a large dosage, i. e. 1064 mgr.-hours radium element with an additional 1 mm. lead filter. The tonsil has completely disappeared, the fauces is empty and has a smooth mucous membrane of normal colour. Through this treatment the left tonsil was also reduced and, the patient not having had any attack of angina after the first treatment, I cut down the dosage for this tonsil to 120 mgr.-hours. Owing to this, the tonsil has become average-sized, but the patient is completely free from all symptoms of her troubles. No atrophy is to be seen in the throat and the patient is not troubled by any dryness. On the contrary, she considers that she has felt altogether more healthy and strong after the treatment than she has done for many years.

The second patient has been treated according to the usual principles. The tonsils as well as the adenoids have been considerably reduced and the angina attacks have disappeared.

The third patient received treatment only on the left side, as the right one showed no troublesome symptoms. Symptomless.

None of these patients exhibited any abnormal signs during the course of reaction, and no bleeding occurred during that period.

Group XIV. Cases of various complaints remaining after tonsillectomy.

This group comprises three patients who have suffered from recurring attacks of angina.

In one of these patients some small remains of tonsillar tissue were seen lying in the right fauces and infiltrating the posterior pillar. The second patient had also some remains of tonsillar tissue in the posterior pillar. Both of them had troubles from this side with angina symptoms and slight rises of temperature. The third patient exhibited no visible tonsillar remains on either side, but displayed a nice, smooth cicatric, the colour of the mucous membrane with normally shaped faucial pillars. He complained, however, of a feeling of tenseness and fulness on the left side, and an attempted treatment was therefore made. The troubles persisting, I managed to probe a small remaining crypt, the opening of which I had not been able to probe before. This crypt was excised, and the patient became free from symptoms.

Group XV. Cases of unilateral tonsillar hypertrophy, causing obstinate vomitings.

To this group I have referred one patient who I have not been able to range in any of the other groups. The patient suffers from an achylia gastrica with periodical vomitings. She has been treated by a prominent physician who considered that the vomitings were possibly due to the enlarged palatine tonsil of the right side. The patient herself was of the definite opinion that the nausea came from that place. She did not suffer from any pronounced angina troubles.

On eximination the right tonsil was found to be moderately enlarged. The patient received radium treatment on that side with 288 mgr.-hours radium element. The tonsil was reduced to normal size, and the patient has no longer been troubled by vomitings.

Summary of the results obtained by the Radium treatment.

Out of the 150 patients who have come for radium treatment, 135 may be acknowledged symptomless as far as the chronic tonsillitis is concerned. Five out of these 135 patients still have some secondary symptoms. In four cases the treatment has had no visible effect on tuberculous lymphomata (group X), and in one case there still remain some neuralgic pains.

Eight patients have not been completely freed from symptoms as far as their throat troubles are concerned, but three of them have taken only a partial treatment. (Two in group I and one in group XIV). Two of the others are, howewer, much improved (group I), inasmuch as they have each had only one attack of an-

gina. Only three have had attacks of angina to the same extent as before. (One in group I and two in group IV).

The treatment has been abandoned i 7 cases. I stopped the treatment in three of these cases as there was no indication as to the advantage of continuing it.

Discussion on the size of the dosage.

1. If I treat the skin of the ventral side of the forearm over an area of 25×10 m. m. with the same dosage and the same technic as I generally use for a tonsil of the same area, then I get a reaction which is similar to the common radium reaction of the skin, but which differs from the reaction of the tonsil. While the reaction after the treatment of the tonsil passes off in about 20 days, the reaction of the skin takes a longer time.

The first 14 days show only a very faint redness of the skin. This redness becomes intensified during the 3rd and 4th week and a small edema appears, which makes the treated surface puffy. The edema increases during the 5th week, at the end of which the superficial layers of the epithelium are desquamated, whereby a superficial ulceration appears. After 4 or 5 days the healing begins from the edges and the lesion is completely healed after 10 to 12 days. The reaction takes about 7 weeks before it has subsided entirely. The treatment leaves a pale, soft scar on the level of the skin and an inconsiderable pigmentation of the surrounding surface.

The reaction upon the tonsil has a shorter and quite different course, partly owing to the anatomical structure of the mucous membrane and partly to the property of the tonsillar tissue to react against external influences by means of a fibrinous transudation in and beneath the epithelium with a formation of a fibrinous membrane. Whilst in the skin reaction the subepithelial transudation is comparatively insignificant and manifests itself as a slight swelling of the treated area, the transudation in and beneath the tonsillar epithelium becomes particularly great already on the 9th or 10th day after the treatment and results in an early desquamation of the superficial layer of epithelium. A rapid epithelialization now takes place beneath the membrane and from the edges of the surrounding mucous membrane, so that the subjacent surface is covered with a new epithelium already when the membrane is resorbed.

The superficial reaction of the tonsillar tissue is, thus, entirely completed on the 20th day after the commencement of the treatment, when a skin reaction is still at its climax.

2. If I increase the distance to 3 m. m. in the skin treatment, then I get a far milder reaction with a faint redness in the 4th or 5th week, which redness lasts for a fortnight and is succeeded by an inconsiderable desquamation of the most superficial epithelial layers.

If, when treating the tonsil, I increase the distance to 3 m. m. by interposing an extra layer of plastic mass, 2 m. m. thick, the superficial reaction with membrane formation and desquamation of the epithelium does not set in, but I get only a faint redness on the surface, followed by a more or less marked decrease in the size of the tonsil.

The course of reaction on the skin as well as on the mucous membrane in a treatment given at a distance of 1 and 3 m. m. shows, thus, that the radiation which produces this superficial reaction with membrane formation and epithelial desquamation, is considerably diminished in the most superficial 2 m. m. of the tonsillar tissue, which of course may be expected both on account of the difference in distance and of the absorption of the soft rays. This soft radiation, producing the surface effect, as well as the soft γ-radiation, consists of a secondary β-radiation from the filter and from the thin layer of plastic mass.

3. The American authors who practise radium treatment, employ a very soft primary radiation, as they use flat dermatological containers from which 50 % or more of the primary β-radiation reaches the superficial layers of the tonsil. They have a dosage of 30—50 m. gr.-hours radiumelement and thereby get a reaction which »turns the part white». Because of this weak filtration only a comparatively small dosage of radium can be used, and most of them must therefore improve the local radium treatment by an external roentgen treatment.

4. When determining the dosage I have taken as granted that the chronic tonsillitis is caused by an infection from the surface of the tonsils and the crypts, except of course septic metastasis from some other demonstrable focus of infection. The tonsillitis must therefore be regarded almost as a local affection, whether there is only an inconsiderable local infection of one of the palatine tonsils or the infection affects the entire ring of WALDEYER.

DIETRICH emphasizes particularly the superfical infection as being the entrance-gate for the tonsillar inflammation.

I have, thus, tried to produce:

a) *a comparatively strong effect upon the most superficial tissue layers, thereby causing a resorption of these which in their morbid condition con-*

stitute an easily accessible point of entrance for infections. By this means trying to effect the surface reaction obtained by the American authors.

b) *a comparatively powerful deep effect, able to reduce the tonsils to a normal size by causing an atrophy of the hypertrophied portions.*

By combining a soft surface radiation consisting of the soft γ-radiation and a secondary β-radiation from the filter and the thin layer of plastic mass, with a harder radiation, consisting of penetrating hard γ-radiation, I have managed to obtain a powerful superficial as well as deep effect, thereby avoiding a combination of radium and roentgen treatment.

5. On collation of the results obtained by the treatment it appears (Fig. 6. Pag. 22) that everyone of the five patients treated with a dosage exceeding 288 m. gr.-hours have been completely symptomless. On comparison of the results obtained in the patients treated with the average dosage and the small dosage, the fact becomes apparent that a somewhat poorer result has been obtained by the smaller dosage.

Thus, 45 patients out of the 47 belonging to Group I and treated with 240—288 m. gr.-hours have become symptomless, whilst 2 still have their symptoms. Three patients have been treated with the smaller dosage, out of whom two have become symptomless and one not.

6. The results of treatment are remarkable in the cases who have only been partially treated. All symptoms from the treated side have disappeared in five of these cases, whereas they are still present on the other side, although much less than before the treatment. In 18 cases there was nothing to indicate a treatment of more than one tonsil, as the other tonsil did not give any trouble and was not very much enlarged. The remaining 17 cases have become completely free from symptoms by treatment of only one tonsil.

7. ALBANUS states that a painting of the mucous membrane with cocain-adrenalin solution produces anemia of the membrane to such an extent that its thickness is reduced from 7 to 4 m. m., and he further emphasizes the consequence of this fact for the deep dosage. REICHER and LENZ have pointed out that an adrenalin-anemia, produced electrophoretically, almost doubles the normal tolerance of the mucous membrane. The anemic tissue absorbes a lesser quantity of rays than the normal tissue. They emphasize, however, that this effect is produced only by a strong anemization. FREUND has had an experience quite the reverse of this, having found an increased sensibility in connection with subcutaneous injection of adrenalin.

I have made comparative investigations concerning the course of reaction in the mucous membrane of the mouth which has been painted with a 20 % solution of cocain plus a few drops of adrenalin, and which has not been treated in this manner. I have not been able to find any clinical difference at all between the two courses of reaction, either with regard to intensity or duration.

But on the other hand I have noticed a quite distinctly increased sensibility of the mucous membrane in the few cases that I have treated in immediate connection with an angina, when the symptoms of irritation were still present.

Eventual injuries by the radium treatment.

The reactionary course after the treatment shows that the dosage does not cause any hitherto observed injury. The period of observation having been altogether too short to exclude to a certainty delayed injuries in the form of late atrophies, I have made a post-examination of some of the patients, whose tonsils have been treated, during 1916—1919.

1) Female, aged 46, sarcoma tonsillae, treated in Nov. 1916 with 616 m. gr.-hours Radiumelement without any additional filter, in Dec. 272 m. gr.-hours with an additional lead filter of 1 m. m., and in May 1917 with 600 m. gr.-hours, with an additional lead filter of 1 m. m.; living, symptomless, no atrophy of the pharyngeal mucous membrane, no trouble.

2) Female, aged 50, sarcoma tonsillae, treated in Sept. 1919, 735 m. gr.-hours Radiumelement without filter; living, symptomless, no atrophy and no subjective troubles.

3) Male, aged 30, sarcoma tonsillae, treated in July 1919, 960 m. gr.-hours Radiumelement; living, symptomless without any subjective troubles.

4) Male, aged 56, sarcoma tonsillae, treated in June 1919 with 844 m. gr.-hours Radiumelement and in July of the same year with 285 m. gr.-hours, living, symptomless, no subjective troubles.

The power of filtration of the tubewall corresponds to that of one mm. lead.

Besides these I have a number of cases of tonsillar tumours, treated during the last years with a dosage two or three times as large as that employed in chronic tonsillitis, without any injuries having occurred.

Roentgen treatment of chronic tonsillitis.

For estimating the results obtained by roentgen treatment of cases of chronic tonsillitis I have had the following material at my disposal:

I) A few patients treated by me.

II) The results of a post-examination of patients treated for tuberculous lymphomata of the neck.

1. The technic employed in the treatment has been as follows: Distance 23 c. m. Spark-gap 37 c. m. Milliampères 2. Filter 3 m. m. aluminum. The field of incidence at the angle of the jaw 4×7 c. m. Dosage $^1/_4$ HED.[1] 1 or 2 treatments have been given on either side in each series with an interval of one or two days between each treatment. 3 to 4 series have been given with an interval of two or three weeks. The total dosage has thus been 2—3 HED[1] in 4 months.

2. Neither any subjective nor any objective reaction has been observable after the treatment. The tonsils have decreased somewhat in size and the crypts have become distinctly shallower and the plugs of exudate reduced in number.

3. Four patients have been treated who all suffered from monthly attacks of angina. One of them got his usual attack immediately after the treatment, for which reason he ceased the treatment and chose surgical tonsillectomy in order to get rid of his troubles.

One patient got her usual angina after the first serie, though it was very much milder than the the previous ones. Since then she has had no attack of angina.

A third patient has had three series and has not had a single attack of angina since the commencement of the treatment. She had very large tonsils which have decreased comparatively sligthly. (Fig. 31. Pag. 31.)

These three patients were treated in the autumn of 1922. The fourth patient was treated in 1921. Besides the ordinary treatment from without, he got roentgen treatment with lead-glass tube through the mouth and directly against the tonsils, altogether $^1/_3$ HED on either tonsil. The tonsils have not decreased in size anything worth mentioning, but the patient has not had any attacks of angina since the treatment.

It is a characteristic feature of these cases that the attacks of angina have disappeared after a couple of series of treatment, wheras

[1] »Hauteinheitsdosis» of Wintz.

the tonsils have not been very much reduced in size in comparison to the diminution generally obtained after a radium treatment.

II. I have further made a postexamination of 150 patients, who had been treated for tuberculous lymphomata of the neck in the Seraphimer-Hospital in Stockholm during the period of 1911—1921.

24 of them or 18 % have had attacks of angina, whereas the remaining 126 or 82 % have not had angina after the beginning of the treatment.

This fact agrees well with the result of the examination VAN ALLEN made of the material at his disposal, where 80 % have been relieved of their angina during the course of roentgentreatment for tuberculous lymphomata, whilst 20 % have had attacks of anginas, although in a milder form than before the treatment.

III. Thus, our experience goes in the same direction as the French and the American one. The roentgen treatment takes however, far longer time than the radium treatment, as according to the American technic one must average 6 to 8 series of treatment with an interval of 2 weeks between each treatment, making altogether a time of treatment of 3 to 4 months. The favourable results seem to appear only after a couple of series of treatment. The diminution of the tonsil is not so evident as by the radiumtreatment.

The effect of the radiological treatment on other organs.

1. During and after the treatment, examinations have been made of the pulse frequency, blood pressure and temperature. At the commencement of the treatment the pulse generally increases to about 120 but is normal again after about half an hour. The blood pressure does not vary during the treatment, and the temperature is not influenced by the course of reaction.

2. Careful blood examinations have been made on several patients. The blood tests have been taken on an empty stomach at 8 a. m. or, if this has been impossible, at times which have been constant for every patient, so as to avoid the physiological day variations in the blood formula. They were all taken from the lobe of the ear with the aid of Francke's needle, and the blood was examined for erythrocytes, hemoglobin and leucocytes. A smear was prepared every time and was coloured with Giemsa's solution. At the differential counting 300 blood corpuscles were counted.

3. The blood changes after roentgen- and radiumtreatment have been very closely studied during the last fifteen years by the investigations of HEINECKE, LINSER and HELBER, KRAUSE, BENJAMIN, VON REUSS,

SLUKA and SCHWARTZ, AUBERTIN, BEAUJARD, WERTHEIMER, SIEGEL, WESTMAN and others.

My examinations show principally the same course of reaction as is found in patients who still have their power of reaction and who have been treated with a castration dosage, i. e. a reaction of the first degree, according to WERTHEIMER and SIEGEL.

Already during the first hour of the treatment there is generally a pronounced hyperleucocytosis, the number of the leucocytes becoming sometimes double to what it is at the commencement of the treatment. As a rule this leucocytosis continues during the course of the treatment. The hyperleucocytosis begins in rare cases one or two hours after the treatment is finished. The radium tubes having been removed, the blood picture very soon becomes normal, so that it has the same quantitative and qualitative composition as before the treatment, already some hours after the treatment. (Fig. 32.)

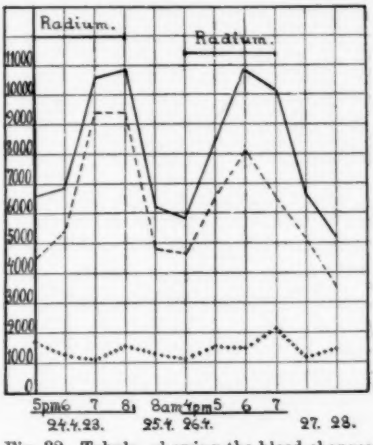


Fig. 32. Tabula, showing the blood changes in the same patient during the course of treatment. Only one day between the two treatments. The unbroken line gives the number of leucocytes, the dotted line the number of neutrophil polynuclears, and the crossed line the number of lymphocytes.

The hyperleucocytosis consists during the treatment of a pronounced absolute neutrophilia with a relative lymphopenia.

During the next few days a relative lymphocytosis together with a small decrease in the number of the leucocytes appears in some patients. No marked leucopenia occurs, and the blood picture of most patients is quite normal.

This blood reaction agrees well with the supposition that the primary leucocytosis is due to an influence on the circulating blood (BENJAMIN, VON REUSS, SLUKA and SCHWARTZ), whereas the subsequent leucopenia is thought to be caused by a radiation effect on the blood-forming organs, the functions of which would thereby be abated.

On the other hand one notices a small increase of the neutrophil leucocytes in a number of patients on the 9th or 10th day, whereas the number of lymphocytes remains normal. This top on the curve of the neutrophils coincides apparently as to time with the reaction which sets in on the tonsil, forming a membrane on the surface. After a couple of days the blood picture has again become normal.

(Fig. 33.) Similar changes are observed during the treatment of the second tonsil and of the pharyngeal tonsil.

4. HASSELBALCH and HEYERDAHL, ELLERMANN and ERLANDSEN have demonstrated how extremely sensitive the leucocytes are to any influence from external causes. Thus, the number of leucocytes may alter when one changes from a recumbent to a standing position (the so-called static leucocyte reaction). Also psychical influences have a very great effect on the blood picture. HEYERDAHL considers that these variations are not due to any changes in the frequency of the pulse but to changes in the difference between the systolic and diastolic blood pressure.

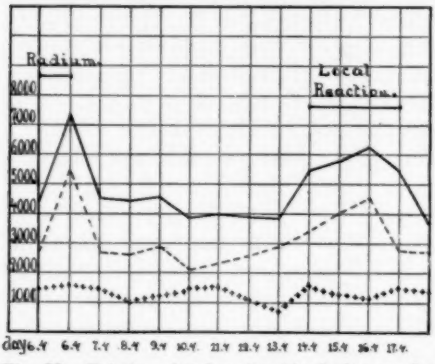


Fig. 33. Tabula, showing the blood changes during the radium treatment and during the next 12 days. The significations are the same as in Fig. 32.

In his critical enquiry into the blood examinations of RUSS, LEITSCH has pointed out that even the psychical anxiety alone, which is associated with a roentgen treatment, may produce blood changes that very much remind of those caused by the roentgentreatment itself, and he has obtained similar blood changes in animals only by performing a feigned radiation of these animals.

5. For this reason I have made the following control experiments, the results of which are recorded in the form of curves in fig. 34.

One patient had to fast from the morning until 4 p. m. The blood tests that were made during this time, did not show any characteristic changes.

On the next day the pharynx was anesthetized with cocain, the dental plate was fixed against the teeth and the inner arm of the tongs was introduced, though it was not applied against the tonsil, but laid free in the mouth. The patient had this arrangement on for three hours without any charateristic changes appearing in the blood picture.

On the third day a feigned treatment was given, the inner arm of the tongs being applied to the tonsil with the usual pressure but without any radium. A distinct increase in the number of the neutrophil leucocytes occurred then. The increase was at its height a couple of hours after the treatment. Next morning the blood picture was again normal.

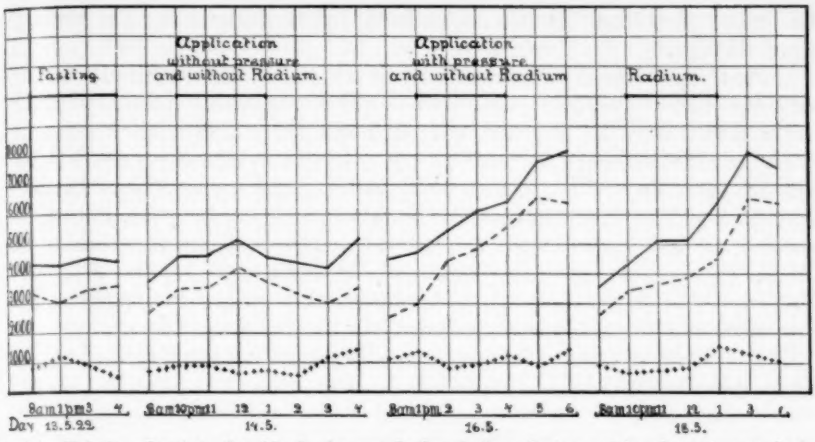


Fig. 34. Tabula, showing the blood changes during fasting, during a feigned treatment, during a treatment with pressure alone against the tonsil and during a regular radium treatment. Signification as in fig. 32.

This experiment proves that the blood change in this case must be due to some other cause than the radium treatment. The two eventualities that can be taken into consideration are either that this reaction is due to the psychical anxiety of the patient for the treatment or that the actual pressure against the tonsil is able to produce the blood change.

Finally, on the fourth day an ordinary radium treatment was given, which resulted in a reaction from the blood almost identical to that produced when pressure was applied to the tonsil without any radium.

In one or two cases I have feigned to treat the patients for an hour and only by pressing against the tonsil. I have then obtained a slight increase in the number of the leucocytes, whilst the usual increase was distinctly apparent at the subsequent radium treatment.

At a control treatment with radium on the ventral side of the forearm, when every psychical influence as well as every influence by pressure can be excluded to a certainty, the usual blood reaction with hyperleucocytosis and lymphopenia sets in. At a feigned treatment of the tonsil the same patient showed, however, an increase of white blood corpuscles from 8 000 to 15 000.

At a control experiment on a woman, aged 65, whose tonsil exhibited a complete senile atrophy, I did not find any effect at all on the blood picture by treating with pressure of the tongs without any radium, whereas a treatment with pressure and radium showed

the same reaction as that obtained by treating the skin with the same dosage.

From the blood examinations it thus becomes evident that the patients after the treatment get a quickly passing primary neutrophilia together with a relative lymphopenia, which is then succeeded by a normal blood picture or by a slight lymphocytosis. On the 9th or 10th day a leucocytosis of short duration sets in, consisting of neutrophilia with a normal number of lymphocytes. This blood reaction is associated with the simultaneous radium reaction on the tonsil. *The short primary neutrophilia seems to be due to a direct effect on the blood corpuscles, a destruction of the lymphocytes being then probably the essential condition for the neutrophilia.*

The blood picture does not experience any permanent change by the treatment.

The effect of the radium treatment on the presence of bacteria.

By the close bacteriological investigations of the American authors it has been proved that the pathogenic bacteria, and especially the hemolytic streptococci, disappear during the course of the roentgen treatment owing to the improved drainage of the crypts.

Unfortunately I have not examined my material with regard to the presence of bacteria, but the present series of investigations into this question argues in favour of similar results being obtained by the radium treatment.

Microscopic examinations of radium treated tonsils.

1. Since HEINECKE in 1903, 1904 and 1905 published his classical investigations on the effect of the roentgen rays on the internal organs and proved the sensibility of the lymphocytes to the roentgen radiation, his observations have been corroborated by a number of authors, for instance the Swede RUDBERG, HELBER and LINSER, KRAUSE and ZIEGLER, AUBERTIN and BEAUJARD, EGGERS, PIGACHE and BECLÈRE, REGAUD and CREMIEU and others.

HEINECKE proved that pathological processes began to appear in the centres of the secondary follicles already after 2 or 3 hours of radiation. Numerous small structureless lumps of chromatin of various forms and sizes were found there, indicating a degeneration of lymphocytes. The fragmentary nuclei were first lying free, but at

the end of two hours large phagocytes appeared and ingested these nuclei. The degeneration and the resorption of its products is completed already after 24 hours. There are then no longer any lymphocytes in the centres of the follicles, but large epithelium-like cells with a round, large nucleus, poor in chromatin. These cells arrange themselves concentrically like the coats of an onion, the whole follicle strongly resembling a cancroid pearl. They are, according to HEINECKE, the normal mesoblastic reticulum cells which are quite covered by the lymphocytes under ordinary circumstances.

After these degenerative processes in the follicles, resulting in disappearance of the lymphocytic elements, a rapid regeneration of the lymphoid tissue sets in, however, also in cases where the large dosages of HEINECKE have been used, so that lymphocytes begin to appear already on the 4th or 5th day. The regeneration is as a rule complete already at the end of four weeks, the lymphoid tissue having then regained its normal appearance.

2. At this point the animals which were used for the experiments, used to die in connection with the ulcerous skin reaction which then appeared. HEINICKE could not explain the cause of death. This was not, according to him, to be found in a change in the lymphoid tissue.

The investigations of FABRICIUS-MÖLLER have lately shed a new light on this question. He has proved that the megakaryocytes of the bone marrow decrease considerably in number, whereby a marked thrombopenia arises, as the thrombocytes are formed from the megakaryocytes. Owing to the pronounced thrombopeni a hemorrhagic diathesis arises with frequent bleedings in various organs, which bleedings are the cause of death.

3. In my investigations on the microscopic changes in the radium-treated tonsils I have had for my material tonsils which have been extirpated a certain time after the treatment. The tonsils have been fixed and stained by Ellermann's method which gives a very beautiful colouring of the nuclear structures.

In a tonsil which has been treated with the ordinary dosage and been extirpated 7 1/2 hours after the commencement of the treatment, I can see extensive changes in the secondary follicles, which changes fully coincide with the pictures of the lymphocytic degeneration obtained by HEINECKE and RUDBERG. (Fig. 43. Tabula XI.)

The centres of the secondary follicles appear as usually clearer than the peripheral zone, but over the whole of the centre there lies as it were, a fine dust of darkcoloured particles. One notices here all the phases of degeneration of the lymphocytes. Several lymphocytes exhibit a nucleus consisting of one single baked lump

of chromatin without any nuclear structure at all and without any membrane, but still having a zone of protoplasma all round it, which points to a pycnotic degeneration of the nucleus. The pycnotic nucleus then becomes irregular and indented and breaks up, forming rounded, semilunar or irregular, large or small lumps of chromatin which are often accumulated into heaps. In several places one can find how these heaps of chromatin globules are lying within large phagocytic cells. The phagocytic cells seem to be reticulum cells liberated from the rest of the reticulum. The small, strong basophile coloured lumps of chromatin soon lose their strong stain, become finally eosinophil and disappear gradually from the follicles. The phagocytes disappear simultaneously.

The peripheral zone round the disintegrating centrum consists of numerous lymphocytes which exhibit a normal nuclear structure. Some of them have, however, a distinct pycnotically changed nucleus.

The next phase that I have had the opportunity to examine was a tonsil which was extirpated 50 hours after the commencement of the treatment. (Fig. 44. Tabula XII.) No sign at all is found here of the intense destruction. In the centre of the follicles there are now no longer any cells of the lymphocytic or lymphoblastic kind, but the cells are large, with eosinophil protoplasm and have a large, oval nucleus which is very faintly stained. These cells are arranged in concentric layers and are apparently reticulum cells which have not come out distinctly before, as they have been covered by lymphocytes.

The lymphocytes in the peripheral zone are now showing pycnotic changes to a far greater extent than in the previous phase, at least one half or two-thirds of the cells have a pycnotic nucleus, the peripheral zone has grown narrower and the cells are not so numerous either. Through these cells one can discern the reticulum cells, which are also arranged concentrically.

A pronounced hyperemia is present in the whole of the tonsil, the blood vessels being over-crowded with blood corpuscles. Crowded blood vessels may also be seen within the follicles.

In specimens obtained from an experimental series of radium-treated rabbit tonsils I find similar changes in the secondary follicles 2 days after the end of the treatment. The centre is void of lymphocytes, the cells in the centre are large with a large nucleus. The protoplasm is filled with eosinophil granules. No giant cells are to be seen in the centre, but in the periphery of the follicles, where pycnotic nuclei are found, there are numerous macrophages.

In fig. 46 Tabula XIII is seen a crypt from a tonsil which has been extirpated 50 hours after the radium treatment. Numerous se-

condary follicles are seen lying along the surface. All of them exhibit centres which are quite void of lymphocytes.

In tonsils ectomized about 20 days after the radium treatment I get about the following picture: (Fig. 46, Tabula XIV). The secondary follicles are still distinctly visible, but some of them are smaller than before and show degenerative changes, while others exhibit the same structure as before. The central clear portion is partly broken up, showing gaps in the tissue. Right in the middle of such a centre one finds often heaps of lymphocytes with normal nuclear structure, apparently immigrated or newformed lymphocytes.

The reticulum cells are not so large, but more like the connective tissue cells. The peripheral zone of lymphocytes has disappeared in a great number of the follicles, and one finds frequently a ring of tightly packed leucocytes in its place. Obviously these are secondary follicles being resorbed.

The rest of the tissue is characterized by a certain degree of lymphopenia, as one is able to distinguish the reticular stroma much more distinctly than before. The lymphocytes present do not, however, exhibit any extensive pycnotic changes. This phase, on the 20th day after the treatment, corresponds to the point of time when the radium reaction generally is almost completed and when the diminution of the tonsil is evident.

This also corresponds to the pictures, which are described by Goerke and Levinstein at the involution of tonsillar tissue.

I have not had the opportunity to study in detail the histological processes in human tonsils during the involution after the radium treatment, nor the structure of the tissue after the reaction is ended, as for quite natural reasons it is not easy to procure material for an investigation of this kind.

4. The histological picture corresponds to the pictures described by Heinecke, Rudberg, Thies and others. These degeneration processes do not exhibit any characteristic features that might be interpreted as specific reactions against the radium treatment. The lymphocyte-free centre in the secondary follicles which makes its appearance after 2 days, has, as mentioned above, been observed in quite a number of infections such as poliomyelitis, diphteria etc. The macrophages are also a regularly occuring feature in the chronically inflamed tonsils. Only the circumstance that these changes appear so regularly and, in different patients within about the same time after the treatment, indicates that they are a consequence of the radium treatment.

SUMMARY

1) The material comprises 154 patients, 150 of whom have been treated with radium and 4 with roentgen.

2 a) The application on the palatine tonsils has been done by means of a tongslike instrument which admits of a minute focussing on the area of treatment as well as of a secure fixation.

b) The application on the pharyngeal tonsil has been done by means of an arrangement which well protects the soft palate and at the same time brings the radium preparation into direct contact with the surface to be treated.

c) Distance treatment has been given by means of the apparatus of Doctor LYSHOLM.

3 a) Very large, firm, fibrous tonsils of grown-up people have been treated with a dosage of about 96 m. gr. Ra. El. for 10—12 hours. The total filtration power of the filter corresponds to the filtration power of 2 m. m. lead. Distance 4—5 m. m.

b) Large soft tonsils of grown-up people and children have been treated with a dosage of 96 m. gr., Ra. El. for $2^1/_2$—3 hours. Filter 0,35 m. m. gold + 0,30 m. m. platinum, corresponding to a filtration power of 1 m. m. lead. Distance: 1 m. m.

c) Small tonsils have been treated with a dosage of about 75 m. gr. Ra. El. for 3 hours by the same technic as in b.

d) The pharyngeal tonsil has been treated with a dosage of 50—96 m. gr. Ra. El. for 3 hours. Filter: 0,35 m. m. gold + 0,30 m. m. platinum. Distance: 1 m. m.

By the application of these dosages a comparatively powerful superficial effect is produced by the soft γ-radiation and the secondary β-radiation, at the same time as a deep effect is obtained by the hard γ-rays.

e) The distance-treatment has been given from the outside with 3—4 gr.-hours at a distance of 5 c. m. and with a total lead filter of 3 m. m.

f) Each tonsil has as a rule received one treatment at an interval of 4—5 weeks between the treatments of the tonsils.

4) The roentgen treatment has been given in agreement in the main with the technic of WITHERBEE.

5) The Radium treatment has given the following results:

a) The greatly enlarged tonsils have decreased to normal or to smaller size; also firm, fibrous and cicatrized tonsils have reacted by a distinct diminution in size. The fissured surface has become smoother and the deep crypts shallower. The contents of the crypts has diminished or disappeared.

b) The attacks of angina, of which the patients have suffered frequently before the treatment, have ceased and the general condition of the patients has been improved. This latter fact applies especially to children.

c) The secondary symptoms, which appear in connection with the local symptoms, have as a rule been greatly improved, when these secondary

symptoms have not been distinctly chronic or have reached a great development.

d) The results of the treatment have so far been permanent during the time of observation.

e) Out of radium-treated patients 135 have been relieved of their local symptoms; 2 have improved considerably; 3 still have troubles from their throat to the same extent as before; 3 who have only had partial treatment, have also their throat troubles still left; 7 have abandoned treatment.

6 a) Out of 4 patients treated with roentgen 1 has become symptomless, 2 very much improved, and 1 has abandoned treatment.

b) A post-examination of 150 patients who have been treated with roentgen for tuberculous lymphoma, has further shown that the frequency of the angina has been unusually low in these patients after the treatment.

7) By the radiumtreatment a temporary change appears in the blood-picture corresponding to SIEGEL's bloodreaction of the first degree.

8) The microscopic examination shows that the diminution in size is due to a degeneration of lymphocytes and to an involution of the secondary follicles.

The radiumtreatment brings on processes which clinically and anatomically resemble those through which the normal physiological involution of lymphoid tissue takes place.

9) The radiumtreatment given by the described technic has not caused any injuries.

ZUSAMMENFASSUNG

1) Das Material umfasst 154 Patienten, von welchen 150 mit Radium und 4 mit Röntgen behandelt worden sind.

2 a) Die Applikation an den Gaumenmandeln geschah mittels eines zangenähnlichen Instruments, welches sowohl eine genaue Einstellung auf das zu behandelnde Gebiet als auch eine sichere Fixation ermöglicht.

b) Die Applikation an die Rachenmandel geschah mittels einer Anordnung, welche den weichen Gaumen gut schützt und gleichzeitig das Radiumpräparat in direkte Berührung mit der zu behandelnden Oberfläche bringt.

c) Distanzbehandlung wurde mittels des Apparates von Dr. LYSHOLM vorgenommen.

3 a) Sehr grosse, feste, fibröse Tonsillen bei Erwachsenen wurden durch 10—12 Stunden mit einer Dosis von ungefähr 96 mg Ra. El. behandelt. Das Gesamt-Filtrationsvermögen des Filters entspricht dem Filtrationsvermögen von 2 mm Blei. Distanz 4—5 mm.

b) Grosse, weiche Tonsillen bei Erwachsenen und Kindern wurden durch $2^1/_2$—3 Stunden mit einer Dosis von 96 mg Ra. El. behandelt. Filter 0,35 mm Gold + 0,30 mm Platin, dem Filtrationsvermögen von 1 mm Blei entsprechend. Distanz 1 mm.

c) Kleine Tonsillen wurden durch 3 Stunden mit einer Dosis von ungefähr 75 mg Ra. El. und mit derselben Technik wie in Punkt b) behandelt.

d) Die Rachenmandel wurde durch 3 Stunden mit einer Dosis von 50—96 mg Ra. El. behandelt. Filter: 0,35 mm Gold + 0,30 mm Platin. Distanz 1 mm.

Bei Applikation dieser Dosen wird durch die weichen γ-Strahlen und die sekundären β-Strahlen eine verhältnismässig kräftige oberflächliche Wirkung erzeugt, während gleichzeitig durch die harten γ-Strahlen eine Tiefenwirkung erzielt wird.

e) Die Distanzbehandlung wurde von aussen in einer Distanz von 5 cm mit einem Gesamt Bleifilter von 3 mm durch 3—4 Stunden verabreicht.

f) Jede Tonsille für sich wurde in der Regel einer Behandlung unterzogen, mit einem Interwall von 4—5 Wochen zwischen den Behandlungen einer Tonsille und der anderen.

4) Die Röntgenbehandlung wurde im wesentlichen in Übereinstimmung mit der Technik WITHERBEES vorgenommen.

5) Die Radiumbehandlung hat folgende Resultate gegeben:

a) Die stark vergrösserten Tonsillen verkleinerten sich bis zu normaler oder etwas geringerer Dimension; auch feste, fibröse und narbige Mandeln reagierten mit einer deutlichen Volumverminderung. Die gefurchte Oberfläche wurde glatter und die tiefen Krypten seichter. Der Krypteninhalt wurde geringer oder verschwand.

b) Die Anfälle von Anginen, an denen die Patienten vor der Behandlung häufig gelitten hatten, blieben aus und der Allgemeinzustand der Patienten wurde besser. Das letztere gilt besonders von Kindern.

c) Wenn die Sekundärerscheinungen, die in Verbindung mit den Lokalsymptomen auftreten, nicht ausgesprochen chronisch oder besonders stark entwickelt gewesen waren, wurden sie in der Regel erheblich gebessert.

d) Die Behandlungsresultate erwiesen sich soweit durch die ganze Beobachtungszeit als anhaltend.

e) Von den radiumbehandelten Patienten wurden 135 von ihren lokalen Symptomen befreit; bei 2 wurde der Zustand beträchtlich gebessert; 3 haben weiter ebensostarke Beschwerden seiten ihres Rachens als vorher; 3, die nur eine partielle Behandlung genossen hatten, haben auch ihr Rachenleiden behalten; 7 haben die Behandlung abgebrochen.

6 a) Von 4 Patienten, die mit Röntgenstrahlen behandelt worden waren, wurde einer symptomfrei, bei 2 trat eine wesentliche Besserung ein, 1 entzog sich der Behandlung.

b) Eine Nachuntersuchung von 150 Patienten, die wegen tuberkulöser Lymphome mit Röntgenstrahlen behandelt worden waren, hat ferner gezeigt, dass bei diesen Fällen die Frequenz von Anginen nach der Behandlung ungewöhnlich gering war.

7) Im Anschluss an die Radiumbehandlung tritt eine vorübergehende Veränderung des Blutbildes, entsprechend der SIEGELschen Blutreaktion ersten Grades ein.

8) Die mikroskopische Untersuchung zeigt, dass die Grössenabnahme auf einer Lymphozytendegeneration beruht und auf einer Involution der sekundären Follikel.

Die Radiumbehandlung ruft Prozesse hervor, welche klinisch und anatomisch jenen gleichen, durch welche sich die normale physiologische Involution des lymphoiden Gewebes vollzieht.

9) Die Radiumbehandlung hat, in der beschriebenen Technik vorgenommen, keine Schädigungen veranlasst.

RÉSUMÉ

1) Le matériel comprend 154 malades, desquels 150 ont été traités avec du radium et 4 avec des rayons X.

2 a) L'application aux amygdales palatines est faite au moyen d'une espèce de pince qui permet une mise à point précise sur l'endroit à traiter ainsi qu'une fixation solide.

b) L'application à l'amygdale pharyngienne a été faite de façon à bien protéger le palais mou et à mettre en même temps la préparation de radium en contact direct avec la surface à traiter.

c) Le traitement à distance a été donné à l'aide de l'appareil du docteur LYSHOLM.

3 a) Des amygdales très grandes, dures et fibreuses d'adultes ont éte traitées avec une dose d'environ 96 mg d'élément de radium durant 10 à 12 heures. La force de filtration totale du filtre correspond à la force defiltration de plomb de 2 mm. Distance: 4 à 5 mm.

b) Des amygdales grandes et molles chez des adultes et des enfants ont été traitées par une dose de 96 mg d'élément de radium durant $2^1/_2$ à 3 heures. Filtre: or de 0,35 mm + platine de 0,30 mm. correspondant à une force de filtration de plomb de 1 mm. Distance: 1 mm.

c) Des petites amygdales ont été traitées par une dose de 75 mg d'élément de radium durant trois heures d'après la même méthode que sous b.

d) L'amygdale pharyngienne a été traitée par une dose de 50 à 96 mg d'élément de radium durant 3 heures. Filtre: or de 0,35 mm + platine de 0,30 mm. Distance: 1 mm.
En appliquant ces doses un effet superficiel comparativement fort est produit par les rayons γ moux et les rayons β secondaires en même temps qu'un effet profond est obtenu par les rayons γ durs.

e) Le traitement à distance a été donné extérieurement avec 3—4 g-heures à une distance de 5 cm et un filtre de plomb total de 3 mm.

f) Chacune des amygdales a été traitée une fois et l'interval entre les traitements de chaque amygdale a été de 4—5 semaines.

4) Le traitement aux rayons X a principalement été donné d'après la technique de WITHERBEE.

5) Le traitement au radium a donné le résultat suivant:

a) Les amygdales fortement grossies ont diminué jusqu'à leur volume normal ou davantage; aussi des amygdales dures, fibreuses et des amygdales cicatrisées ont réagi par une diminution en volume distincte. La surface qui avait des fissures est devenue plus lisse et les creux moins profonds. Le contenu des creux a diminué ou disparu.

b) Les cas d'angine dont les malades avaient souffert fréquemment avant le traitement ont cessé et l'état général des malades est amélioré. Ce dernier fait se rapporte surtout aux enfants.

c) Les symptomes secondaires, apparaissant en relation avec les symptomes locaux ont en règle subi une amélioration dans les cas où ces symptomes secondaires n'ont pas été distinctement chroniques ou trop avancés.

d) Les résultats du traitement ont été permanents durant le temps d'observation.

e) Des malades traités au radium 135 ont été délivrés des symptomes locaux; 2 montrent une amélioration considérable; 3 ont encore des malaises dans leur gosier comme avant le traitement de même que 3 qui n'ont subi qu'un traitement partiel; 7 ont interrompu le traitement.

6 a) De 4 malades traités avec des rayons X 1 n'a plus de symptomes, 2 se portent mieux et 1 a quitté le traitement.

b) Un examen postérieur de 150 malades traités avec des rayons X pour lymphome tuberculeux a encore montré que les cas d'angine chez ces malades étaient extraordinairement moins fréquents après le traitement.

7) Le traitement au radium a dans sa suite un changement temporaire dans le sang correspondant à la réaction de sang du premier degré de SIEGEL.

8) L'examen microscopique démontre que la diminution en volume est dûe à la dégénération des lymphocytes et à une involution des follicules secondaires.

Le traitement au radium produit des changements qui cliniquement et anatomiquement ressemblent à ceux durant l'involution physiologique normale de tissu lymphoïde.

9) Le traitement au radium fait d'après la technique décrite n'a pas des suites désavantageuses.

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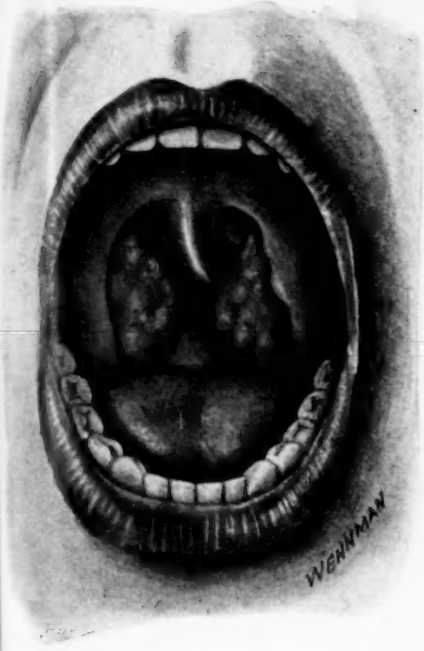
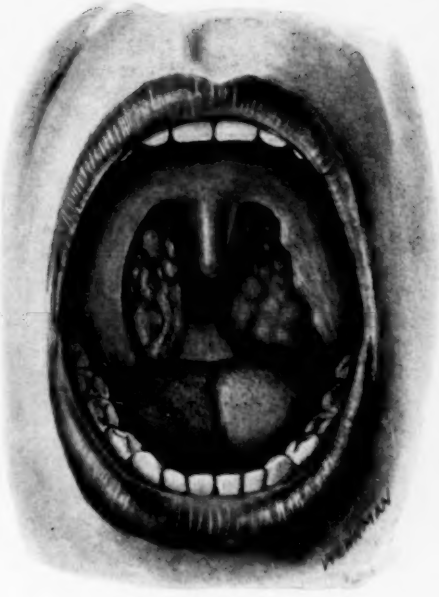


Fig. 35.



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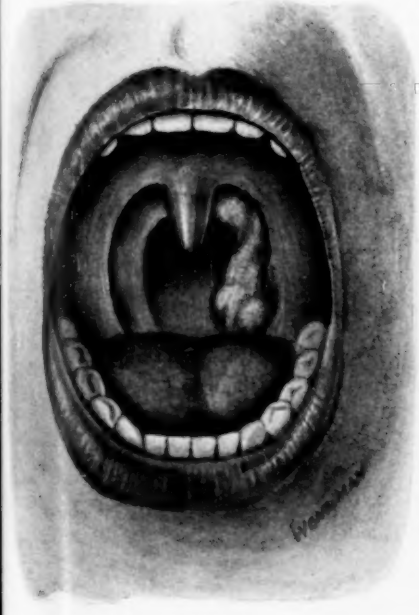


Fig. 37.

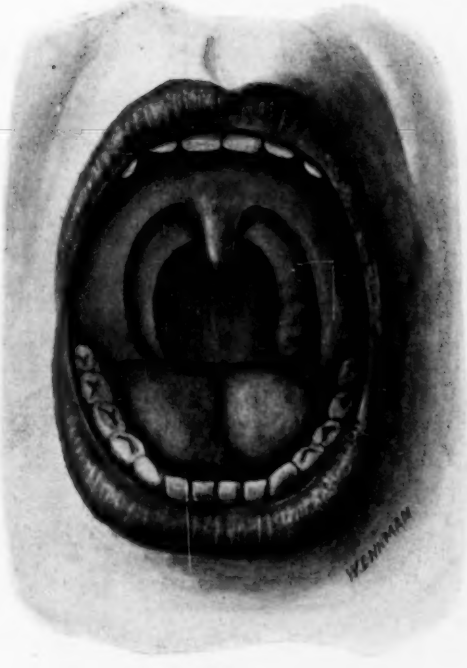
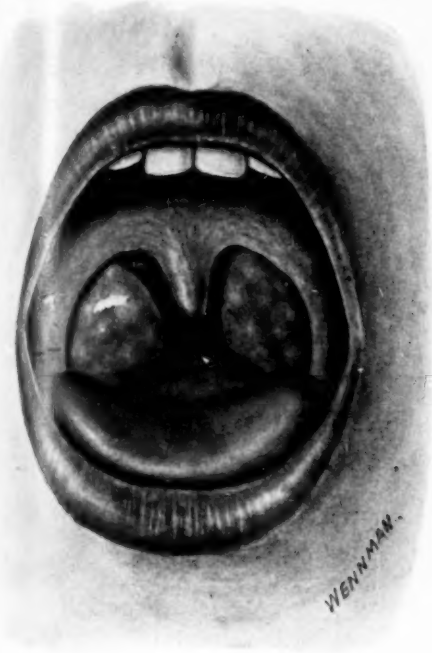


Fig. 38.

Tabula X



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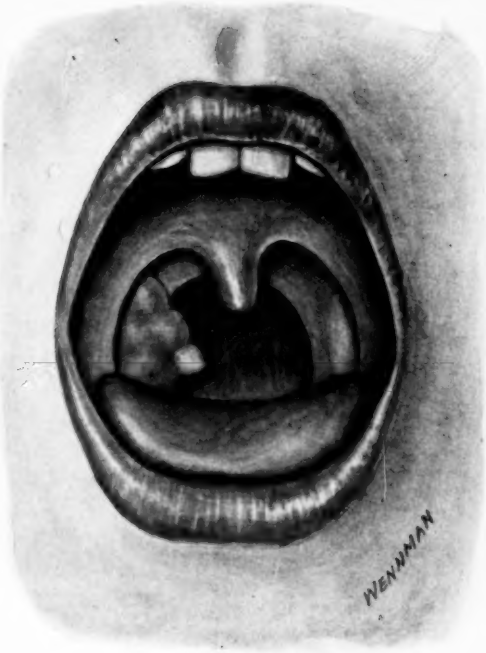

Fig. 40.

Fig. 41.

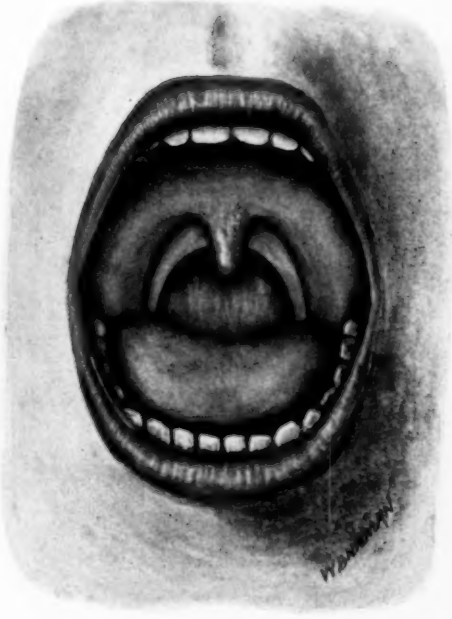

Fig. 42.

Tabula XI

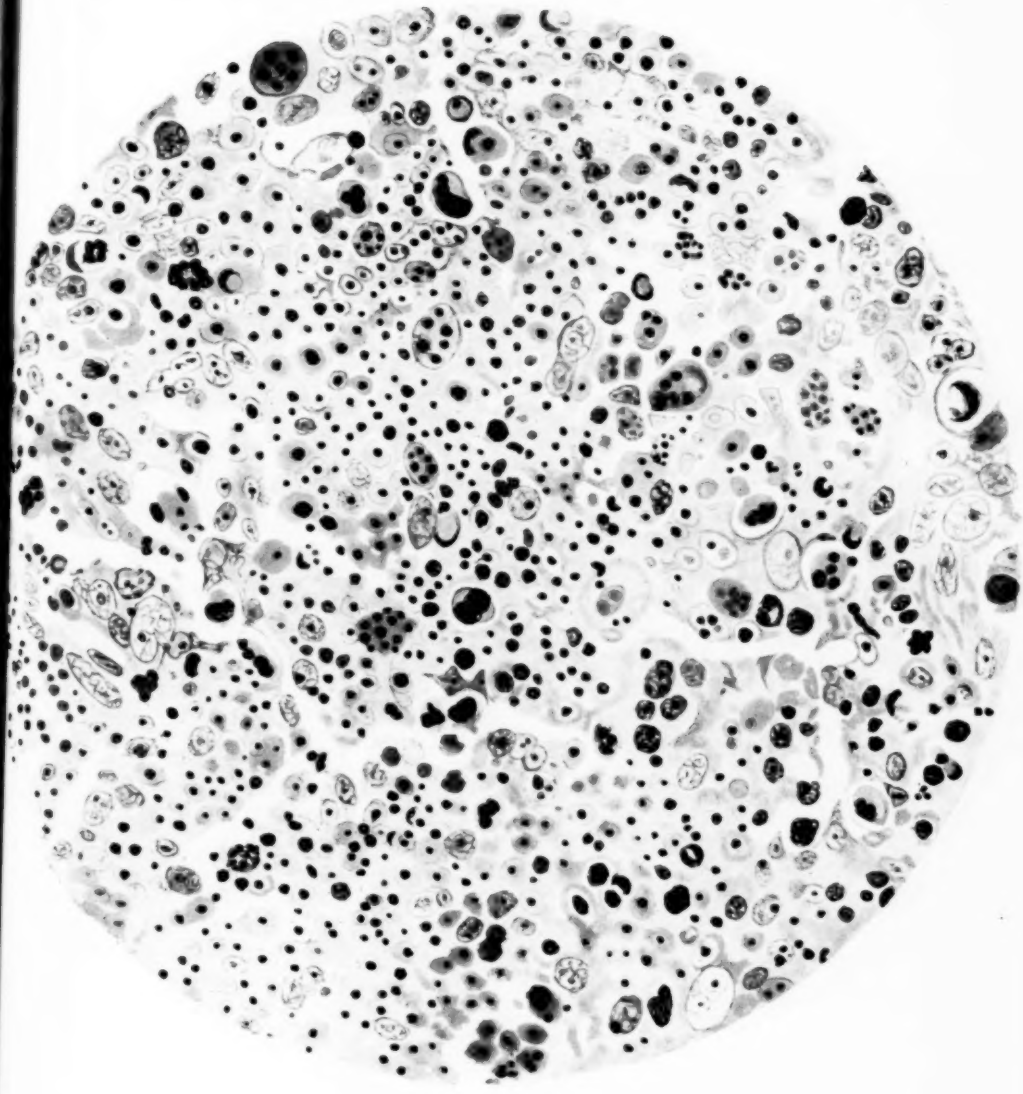

Fig. 43.

Tabula XII

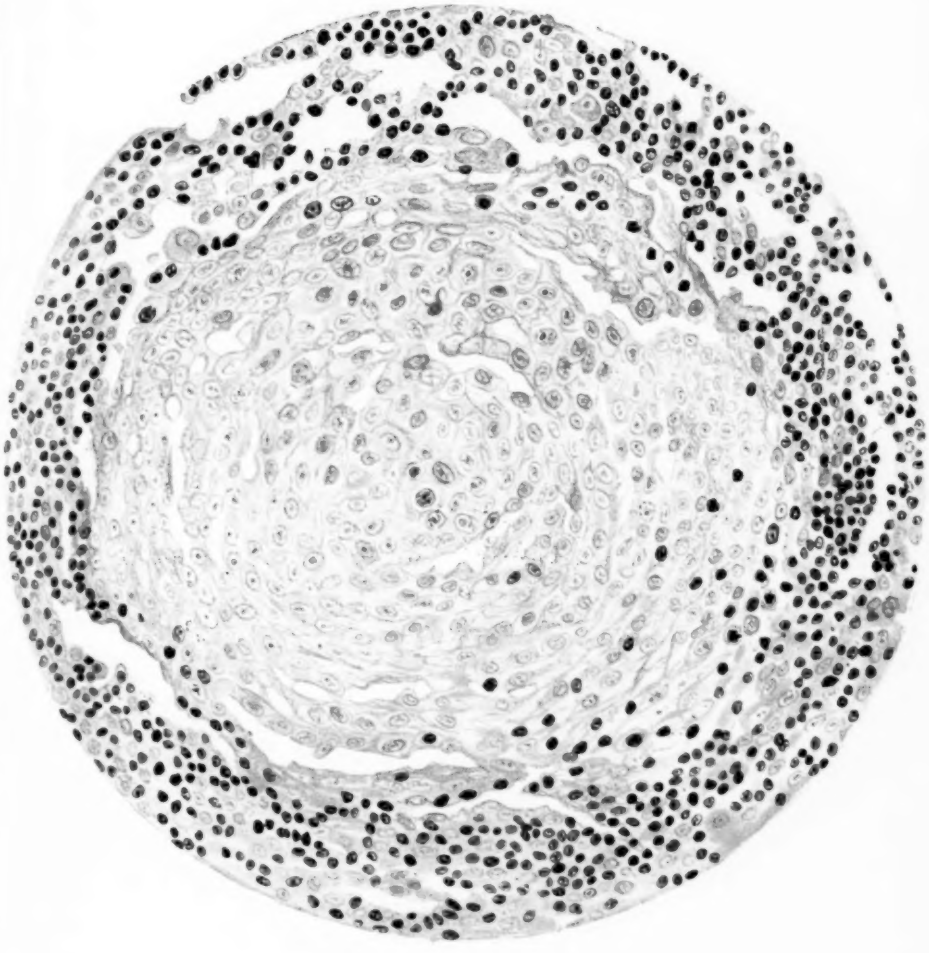

Fig. 44.

Tabula XIII

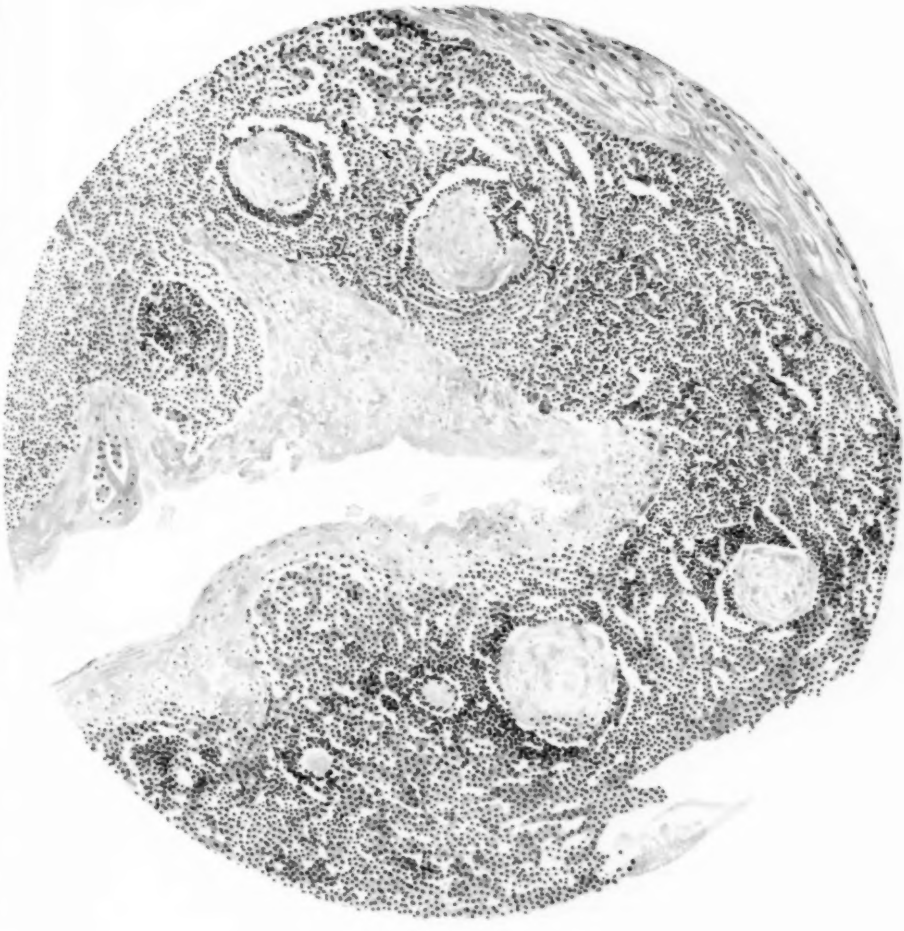

Fig. 45.

Tabula XIV

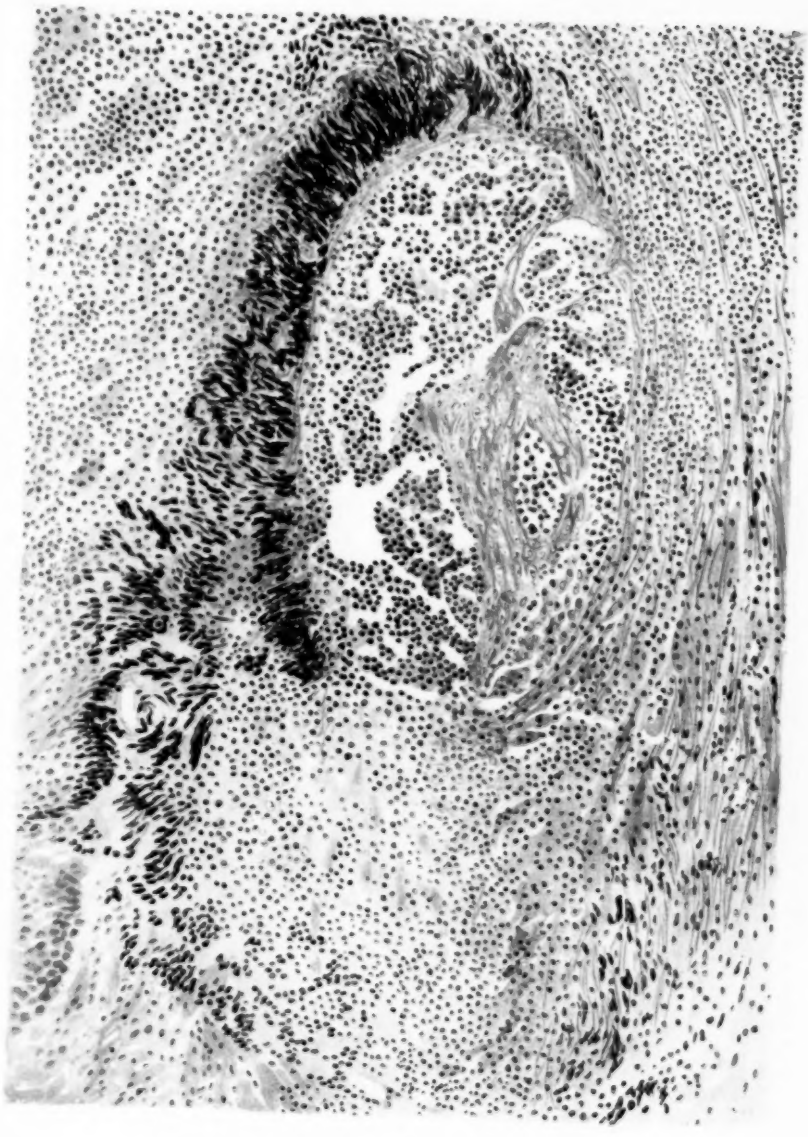

Fig. 46.

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»THE ACUTE BONE ATROPHY» AND ITS ROENTGEN PICTURE

by

Chr. I. Baastrup

(Tabulae XV—XVIII)

Introductory Address at the 3rd Congress of The Northern Association for Medical Radiology in Stockholm June 18th and 19th 1923

At the second meeting of the »Northern Association for Medical Radiology», held in Copenhagen in September 1921, the late Professor J. F. FISHER proposed that the question of the s. c. »acute bone atrophy» should be brought up for discussion in an introductory discourse at the next meeting of the Society, to be held in Stockholm in June 1923.

Professor FISCHER who had a keen eye for the essential part of the medical problems, felt dissatisfied with the not very distinct views prevailing in this important department of medicine.

The proposal of Professor FISCHER won a general support.

In the following I have tried to give an account of the important facts concerning the above-mentioned subject which appeared in the enormously rich literature that had been written about the questions connected with this matter, and I have tried to set forth the conclusions which I have arrived at after having been working at this task.

By »bone atrophy» in a roentgenological sense is meant a pathological change in the bones which manifests itself in the roentgen plate by the bones giving a picture of reduced density, owing to their containing a smaller percentage of lime.

The characteristic feature of the »acute bone atrophy» is that changes set in relatively soon after the causal disease. This, it is

said, can be of many different kinds. A whole string of — generally acute — diseases, especially lesions and inflammations of bones as well as of soft parts, are mentioned in the literature as causing the »acute bone atrophy».

In the »acute bone athrophy» there is, patological-anatomically, an »osteoporosis», i. e. an osteoclastic activity by which bone trabeculæ are gnawed off and partly disappear, and possibly also a »halisteresis», i. e. a loss in the bones of their lime salts.

Therefore it cannot be correct to use the words »osteoporosis» and »halisteresis» as synonymous with »the acute bone atrophy», which is frequently done; »osteoporosis» signifies a pathological process which is found in a number of diseases of the bones, *among which* is the »acute bone atrophy»; »halisteresis» is the denomination of a change in substance found in several diseases of the bones, *among which is possibly* also the »acute bone atrophy».

The expression »ostitis rareficans» which is also found to be used as an expression for the diseases mentioned in this article, is not good either, as there is no reason to look upon these changes as inflammatory. And, further, the term »ostitis rareficans» is used for quite different diseases than the »acute bone atrophy».

In his first article on the »acute bone atrophy» SUDECK calls this disease »acute inflammatory bone atrophy», although he clearly knows that there are no microbes in the »atrophic» bones. KIENBÖCK contests this name. SUDECK acknowledges the views of KIENBÖCK to be correct, and in his following articles he uses the expressions »acute trophoneurotic bone atrophy» or »acute reflex bone atrophy», while KIENBÖCK proposes to name the debated changes »acute bone atrophy», well knowing, however, that the name is not quite satisfactory.

I find the name »acute bone atrophy» to be misleading. By acute is meant (GUTTMANN) »*suddenly beginning, quickly passing*», but the acute bone atrophy sets in by and by, is of rather long duration and passes often into a subchronic stage or even into a permanent state. (In my opinion the word »citus» would have been more felicitous. It means »quick». In the word »citus» lies further the import »hastened on», viz. by the morbid cause which has given rise to the disease).

The name »atrophy» is used in this connection, inter alia, in order to emphasize that the »acute bone atrophy» is a process which is related to the atrophy of skin and muscles that accompanies the »bone atrophy». By »*muscular atrophy*», however, *one understands a diminution in the size of the muscles. A change in the substance of the muscles, is called »degeneration» and not »atrophy»*.

By atrophy is meant »diminution» both in *substance* (eccentric

atrophy) and in *size* (concentric atrophy), but in the course of time this infelicitous double meaning of the word has more and more given way to the meaning *diminution in size* (not, however, in the roentgenology, as I have mentioned before, where the general meaning of the word is »diminution in substance»). The above-mentioned development of the word »atrophy» makes the expression »acute bone atrophy» rather unfortunate, as it is here not a question of any diminution in size of the bones (except in growing individuals or when the process is very lingering, and therefore no »acute» character can possibly be attached to it, fig. 4).

On the whole, the above-mentioned expressions »osteoporosis», »halisteresis», »ostitis rareficans» and »bone atrophy» are employed in many various ways by different authors. Thus SCHUCHARD considers that »eccentric bone atrophy» should be used in connection with the senile and marantic diminution of bone. In the following the capricious use that some other authors make of such words will be referred to now and again. I have tried to use these expressions in the manner which to me seems to be the most explicit and most consequent one.

In order to prevent mistakes I think it would be expedient if »osteoporosis» and »halisteresis» were used *exclusively* to denote the pathologic-anatomical processes which are so aptly expressed by these two words. »Ostitis» ought to be employed only in such cases where there are inflammatory processes. »Bone atrophy» should denote »diminution in the size of the bones» (as seen in stumps, in coxitis in children, etc). The expression »bone degeneration» may be used instead of »bone atrophy» in the meaning »eccentric bone atrophy». If we want to denote the »fatty degeneration» which sets in in cases of eccentric bone atrophy, then we have the expression »lipomacie».

And, finally, the word »rarefication» expresses particularly well and aptly the changes in the bone tissue which is noticed in »acute bone atrophy», »disuse atrophy», »senile bone atrophy» etc. The word strikes me as being felicitous, particularly for expressing the roentgenologically provable changes. (Rarefication means »diminution in substance — chiefly — of bone tissue». If we want to employ a very correct term, then we may speak of »osteorarefication».)

The term »rarefication» may be used in combination with other words, e. g. »rarefication by inactivity», »senile rarefication» etc.

Before a graphic description of the »acute bone atropy» was given on the basis of observations of roentgen pictures, the same phenomena had been set forth several times in the literature. Thus,

BOFINGER and KRAUSE have told us how the bone round a tuberculous focus may become soft and easy to scrape away. Therefore they warn against removing those bone portions which are not affected by the tuberculosis but are only the seat of rarefication. KRAUSE calls the changes »lipomacie», because the intervals between the trabeculæ of the bones are filled with fat. SCHARFF has collected from the literature seven cases of rarefication in connection with acute osteomyelitis, three cases of tuberculosis of the joints and two cases in connection with lesion. SCARFF can himself add one case of all three groups. There is a large work by WILH. ROUX, 1872, on the structure of the bones. He states that in eccentric bone diminution the bone spaces become wider than normal. He distinguishes between two kinds of »atrophy»:

1) atrophy by inactivity, where there is a great resorption of bone trabeculæ, but a hypertrophy of single trabeculæ situated far from each other — a »hypertrophying atrophy», and

2) »simple atrophy», where there are no hypertrophying trabeculæ, but where the bone diminution arises by thick bone trabeculæ getting thinner and large numbers of thin trabeculæ disappearing, whereby the spaces become wider. ROUX bases his statements on examinations of very chronic cases, vix. a stump, a many years old coxitis and a pseudarthrosis.

Later investigators do not seem to found the distinction between »atrophy by inactivity» and other forms of bone diminution on such a pathologic-anatomical basis, but the idea that the »disuse atrophy» is a form apart, as distinct from other forms of »bone atrophy», appears repeatedly in the literature, since ROUX first stated the theory.

FISCHER has, in 1871, given an exellent discription of »trophic changes after nerve lesions in the extremities». Like other authors — especially SAMUEL — he maintains that there exists a »trophic nervous system». The morbid changes after nerve lesions which he has described, may, according to him, be due to unknown »trophic» nerve fibres accompanying the known motor or sensory nerves. In his opinion there can be no question about the vasomotor nerves, because:

1) the changes described by him appear especially when the sensory fibres are damaged, and these are situate' in the posterior nerve roots, while the vasomotor fibres are to be found in the anterior roots.

2) in many cases where the changes described by him are seen, one is not able to point to any signs of disturbances in the functions of the vasomotor nerves: pulse, temperature and secretion.

These arguments are of course of no consequence, and, what is more, FISCHER contradicts himself inasmuch as he has stated earlier

in his work that one may find the said changes after lesion of pure motor nerves, as well as after lesion of pure sensory elements.

I have mentioned FISCHER's work, because his views have in a high degree influenced later works on rarefication.

In 1883 JUL. WOLFF wrote a treatise on »bone atrophy» which has often been quoted. Like FISCHER, he interpreted it as a trophoneurotic disease. He controverts the earlier interpretation of these changes — viz. that they were due partly to inactivity and partly to injuries of the epiphyses.

WOLFF thinks he proves that it cannot be due to inactivity, as the diminution in the bone substance sets in quickly and does not cease when the inactivity ceases. He sees a proof that the process is due to a nervous action and not to a morbid process in the epiphyseal lines in the fact that in cases of coxitis one may find signs of bone atrophy in patients who, for instance, are 30 years old and in whom the epyphyseal lines have consequently disappeared. In coxitis one may also find the part of the extremity situated farthest from the seat of disease — i. e. the foot — to be most diminished. Thus, in this connection he does not distinguish between eccentric and concentric atrophy.

In 1895 STOELTYNER examined microscopically the humerus of a one year old child who had had a fracture of this bone three weeks previously. He found an increase of marrow and a decrease of bone substance, and he considers anemia of the marrow to be the cause of the changes.

Many experiments on animals which throw light upon the question of rarefication, have also been made before the Roentgen rays were discovered, but the results obtained differ often from each other. Only a few of the most important or most discussed results will be mentioned below.

A few days after having cut all the nerves going to one extremity of an animal M. SCHIFF noticed hyperemia of the periosteum and of the marrow, due to vasodilatation. 3 to 6 months after the section of the nerves he found on the operated side a smaller bulk of the bones, wider marrow spaces and thicker periosteum. There were more organic and fewer inorganic constituents than on the sound side. In a dog on which this operation was performed, the bones on the operated side became bendable. Twelve to eighteen months after the operation there was still atrophy, but here and there a greater density was noticed.

MILNE EDWARDS cut the inf. max. nerve of an animal and five weeks later he found a highly developed hypertrophy of the jaw with less organic and more inorganic matter on the side of the lesion

than on the sound side. He got just the reverse results on cutting the limb-nerves.

MANTEGAZZA, 1866, relates how a tendency to pus-formation and necroses and diminution in weight of the bones appears on section of spinal nerves.

LUIGI FASCE, D. AMATO and UGHETTI observed a widening of the marrow spaces, disappearance of the diaphyseal walls and diminution in weight of the bones, due to a decrease of inorganic matter in connection with section of limb-nerves.

KASSOWITZ found after the same operation:

1) considerably less weight, 2) greater flexibility and 3) an increase of the growth in length of the bones on the operated side.

Already on the day after cutting across the nerves DUFOURT found considerable changes in the bones.

NASSE showed that all the substances of the bones are reduced after nerve section, only adipose tissue increases. In young animals the weight could fall to $^1/_3$.

Contrary to many others KAPSAMMER found no diminution of the bones after nerve section, but on the other hand he found dilatation and thickening of the vessels. Similar changes in the arteries were observed by BERVOETS and A. FRÄNKEL in 1896 and by CZYHLARZ and CARL HELBING in 1897. But VULPIAN had mentioned such vascular thickenings already in 1875.

In this connection I may finally mention that in 1899 LAPINSKY proves on the basis of a very great number of works which he quotes, that after nerve section one gets 1) dilatated arteries, 2) slow circulation and 3) higher blood-pressure than normal (yet, some experiments give a somewhat different result).

When the roentgen rays came into use, the conditions for a closer study of the rarefication became better, and, as we have seen before, it was SUDECK and KIENBÖCK who described more systematically the disease called the »acute bone atrophy», in 1899—1900.

The disease is described as follows:

Some weeks after the causal factor a peculiar change in the appearence of the resp. bone is seen in the roentgen picture: *Small areas of reduced density, without sharp contours, are seen in the spongiosa. The bone trabeculæ are »fleecy», the whole picture of the affected part is blurred.* To characterize the appearance such expressions as »dissolved», »spotted», »foggy» or »blotty» are employed. (The »atrophia areata» of CORSON.) (Fig. 1.)

OHLMANN says he has observed a still earlier stage, which manifests itself by small areas of reduced density appearing between the still normal-looking bone trabeculæ.

Later, the so-called second stage begins (the »atrophie en masse» of CORSON). *The blurred appearance has now quite disappeared, the texture stands out clear and sharp with well-defined bone trabeculæ, which are thinner and fewer than normal.* (Fig. 2.)

In the centre of the spongiosa one finds the more or less numerous remains of well-defined, thin and fine spongious trabeculæ, and around these a fine mesh-work is seen, getting finer towards the cortex. The cortex itself has become thinner, as its central layers have got a spongiosa-like appearance. The bone contours stand out very sharply. (Fig. 2.) At an extreme development of the process the spongiosa may be »clear as glass» without any visible spongious structure, yet, by means of a magnifying-glass one may see a mesh-work. The cortex is extremely narrow in such instances. We can find all states of transition between the first »spotted» and the second »clear» stage. (Fig. 3.)

There exists in the literature a great amount of confusion as to the question of the second stage of »acute bone atrophy» or chronic rarefaction (Fig. 4). Several authors do not even enter upon this problem. Others consider the second stage to be the chronic form. SUDECK himself avers that there is no first stage in connection with nervous lesions but includes rarefication after nervous lesions in the »acute bone atrophy». (FINKELNBURG considers he has proved that there exists a »spotted» stage also in connection with nervous lesions.) OHLMANN states that the »spotted» stage is not known in tuberculosis of the bones, but all authors include the rarefication in bone tuberculosis in the »acute bone atrophy». (There is undoubtedly a »spotted» stage in tuberculosis, cf. fig. 5 and COLLIN: Acta Radiologica, vol. I., fasc. 4, pag. 404. figs. 11. 30 etc.) Some authors discriminate apparently in theory between the »acute» and the »chronic» form, as they are using these terms, but as far as I have been able to find in the literature, no one has tried to state any differential-diagnostic symptoms distinguishing between the second stage of the »acute bone atrophy» and the »chronic bone atrophy». Therefore one sees time after time that cases of rarefication, which have been observed years after some lesions or other causal diseases, are referred to under the name of »acute bone atrophy», and KIENBÖCK mentions — arthritis nodosa (!) as one of the causes of the »*acute* bone atrophy». My opinion is that the second stage is identical with the chronic stage, and if the second stage has been reached, then it may continue for years.

Neither is the duration of the stages stated anywhere. SUDECK asserts that the first stage appears 6 to 8 weeks after the causal factor — in one single case he noticed it after 4 1/2 weeks. He does not state any time for the onset of the second stage. EXNER mentions a case of the first stage appearing five months after an acute inflammation, and the second stage nine months after the causal disease. In a case of tuberculosis he saw the second stage three months after the outbreak of the disease. CARL RITTER mentions a rarefication one month after the cause, and a very pronounced reduction in density two months later (it was a case of osteomyelitis acuta). LENK describes a roentgenologically provable rarefication twentyfive days after fracture of the radius. FINKELNBURG has seen a well developed, first stage four to five weeks after gun-shot wounds with nerves lesions. HIRCHMANN and WACHTEL who hawe examined 300 patients with frost-bites and found rarefication of the bones in 80 % of these, claim even to have found the rarefication 14—25 days after the patients had been frost-bitten.

I have not found any mention of how long the »spotted» stage can last. Among my own cases I have two or three times seen the »spotted» rarefication one year after fractures, but otherwise the second stage is always found if there is any rarefication present so long after the lesions.

NASSE noticed reduced density in rabbits two months after section of the nerves, and BRANDES found a distinct rarefication two weeks — in one case one week — after cutting the Achilles tendon. I have seen signs of rarefication in a rabbit one week after bandaging as well as after fracture.

From what has been said above, we find that it is impossible to state even fairly accurately the time when the second stage begins. Firstly, there are many states of transition between the first and the second stage, and secondly there is apparently a considerable difference between the various cases, because of the different causes and rates of development of the process. Many authors claim to have proved that *the changes develop rapidly by traumata and other precipitate or suddenly arisen causes, but more slowly by a less acute cause; moreover, the intensity of the cause is supposed to be a matter of consequence for the rate of development.* On the whole, this is certainly correct, but a quickly developing rarefication is often seen in connection with tuberculosis of the bones, and this is not a sudden cause.

A rarefication but little pronounced may undoubtedly disappear without leaving any marks.

When bones that have been highly rarefied, have once more become sound, the bony structure shows a different picture than nor-

mally: *the spongious trabeculæ are thicker and the interstices wider.* KIENBÖCK is of the opinion that these thick osseous trabeculæ are a compensation arrangement for counteracting the decrease in strength due to the wide interstices. (In this connection one should recall the »hypertrophic atrophy» of ROUX.) KIENBÖCK asserts that only longitudinal trabeculæ remain in a bone thus restored. Even though most of the trabeculæ are now and again found to be lying longitudinally in such bones, many pictures show no absence at all of cross-trabeculæ. But, on the other hand, the *direction of the trabeculæ is often irregular* (cf. fig. 6 and EXNER's pictures in Fortschr. Vol. VI). From the irregular position of the trabeculæ EXNER concludes that new trabeculæ have been formed. The above-mentioned thick trabeculæ point also to a reconstructive activity having taken place during the process of restitution.

The alterations in the bone that have been described above, are found to be most marked in the hands and feet, and here again they are most pronounced in the carpal and tarsal bones and in the bases and capita of the small tubular bones (Fig. 7). On the whole, it is a general rule that the *extremities of long bones are affected at an earlier period and more strongly than the corpora.*

Not only the bony portions situated peripheral to the causal factor become rarefied, but also the *bony portions situated central to the said factor* — at any rate in such cases where the process has lasted for some time and has reached a fairly high development. The process does, however, not follow this rule quite strictly — one may find the tibia rarefied and the fibula practically untouched, or it may have one fragment highly rarefied, while the portions situated central as well as peripheral to the affected fragment show few or no ascertainable changes.

In growing individuals and in processes of long duration one finds, as a rule, the rarefication accompanied by a concentric atrophy.

The authors who have been studying these questions, disagree very much as to how often the rarefication is met with.

THÖLE found it only now and again in old cases of paralysis of the median and ulnar nerves, and OHLMANN states that »acute bone atrophy» is relatively rare. HILGENREINER finds it frequently after gunshot lesions. LENK declares that diaphyseal fractures rarely, metaphyseal fractures often and joint-fractures nearly always result in rarefication. LEHMANN found rarefication »nearly always» in connection with gunshot lesions of nerves, and MANN found it constantly in nervous lesions. My experiments on rabbits seem to

prove that *every kind of severe injuries to the limbs results in a reducea density of bone* in the limb concerned, even though the loss of bone in the less severe cases be not manifest in the roentgen picture, but discoverable only by weighing the bones. A high degree of rarefication is seen only relatively rarely in uncomplicated fractures, but as a rule it is found when the course of the disease is complicated by inflammation or by pseudarthrosis and the like. A high degree of rarefication is nearly always present in tuberculosis of the bones. The »spotted» stage is comparatively rare. Judging from my experience it is found especially in cases where the rarefication sets in rapidly and becomes rather intense.

The pathologic-anatomical changes of the first stage are unknown, and they have probably never been studied at autopsies, except in the above-mentioned case of STOELTYNER. (I have not succeeded in discovering the »spotted» stage during my experiments on rabbits.)

KIENBÖCK declares the picture of the first stage as due to:

1) the photographic plate being comparatively coarse, for which reason all the fine details cannot be recognized,

2) the dissolved lime salts that have not yet left the bone, throwing diffuse shadows which blur the picture of the osseous structure. Moreover, he thinks he is able to infer that it is a question of osteoclastic activity (and possibly of halisteresis).

With regard to the second stage it is here, judging from the entire picture, a question of resorption due to osteoclasts gnawing lacunæ in the bone trabeculæ. Some non-calciferous seams found in some preparations are, according to KIENBÖCK, possibly due to the technic of preparation possibly to some halisteretic elements and possibly to new-formed, not yet calcified trabeculæ. (MALIWA, 1917, asserts that the first stage is due to inflammatory processes, which assertion KIENBÖCK, however, proved to be incorrect, already in 1901.) EXNER found that three specimens of rarefied bones weighed 7 %, 30 % and 67 % resp. less than normal. By burning the bones to ashes and determining the percentage of organic and inorganic matter he found that the *proportions of these two categories were in all three cases about the same as in normal bones — viz. about 60 %, and therefore there can not be any question of halisteresis.* Only in one case he found the amount of lime in the rarefied bone to be about 10 % less than in the normal bone. Therefore, in this case we may perhaps speak about a small halisteresis, or there may have been present some new-formed, not yet calcified elements.

The dissected rarefied bones were soft, brittle and easy to cut with a knife. By squeezing with the fingers the round cross-section of small tubular bones could be moulded into an oval shape. From this he concluded that such bones may bend by loads or by use, and, thus, cause pains. Microscopically, the trabeculæ were found to be fewer and thinner than normal. A number of cells, either resorbing or regenerating, were seen lying against the remaining trabeculæ. OHLMANN examined one case of rarefication in its second stage. The bones were strikingly soft and easily compressible, the diaphyses being, however, comparatively compact. The marrow was thin-flowing, jelly-like and undergoing fatty degeneration. The colour of the marrow was pale red. Microscopically, osteoporosis with osteoclasts was seen.

Simultaneously with the described changes in the bones one finds, as a rule, atrophy of the muscles of the limb or region concerned. The muscular loss is often remarkably great. According to SUDECK, there is a diminished quantitative — not qualitative — reaction on electric currents, galvanic as well as faradic, frequently in a higher degree than what is consistent with the muscular atrophy. CASSIRER states in opposition to this that there is a normal reaction on electric influence.

As a rule, the muscular power is diminished — judging from my own experience it is rather considerably, when the rarefication is well pronounced. RIGLER speaks of a »paralytical weakness of the limb». CASSIRER finds that the muscle power is only diminished a little. And, therefore, he believes that the atrophy affects especially the subcutis and the interstitial connective tissue between the muscles, whereas the muscle fibres are not affected in a corresponding degree. OHLMANN, too, states that on microscopic examination one does not find any loss of muscle fibres.

The skin and subcutis can show considerable changes, especially in cases of strongly pronounced rarefication. The skin feels generally cold which goes to show that the normal regulation of heat by the vasomotor nerves is failing. One finds hard edemata and cyanotic discolouration of the skin, which frequently becomes shiningly atrophic — »glossy skin» — and sometimes scaly. In well-pronounced cases one may find what is generally called »wax-hand». (ERLENMAYER, REMAK and others.) Hypertrichosis may sometimes be seen (REZNICEK and others). The nails may appear longitudinally arched as in drumstick fingers, sometimes with a brown central line that may develop into a diffuse brown discolouration. The subcutis is

atrophic. The venous contour may be absent. Hyperhidrosis is frequently present. The sensibility may be found to be increased, both to pricks and cold (a state of irritation of the sensory nerves). Wounds occur easily and the cicatrization is generally slow.

The function of the joints is frequently very abrogated, active movement is sometimes much inhibited, while passive movement is greatly reduced owing to pains. Pains are further said to be caused by loads. These limitations of movements may vary according as the movements are taking place in various directions, for instance, pronation and supination in the ankle-joint is frequently comparatively more limited than flexion and extension.

The changes may affect the various tissues in unequal degrees in the different cases. As already described by FISCHER, the skin may be intact and the musculature atrophic, or vice versa.

Finally, it must be mentioned that REZNICEK states that he found the vibrating sensation, which is said to be a sign of the sensibility of the bones, to be lessened a few weeks after nervous lesions.

The causes of the »acute bone atrophy» are, as stated, said to be many and various.

In the first group are lesions both of bones and soft parts, and especially lesions affecting joints, even though there be no fracture. Acute inflammations are then mentioned, e. g. phlegmon and osteomyelitis acuta (Fig. 8). Further, tuberculosis of bones and soft parts, and syphilis of bones. (HAHN and DEYCKE-PASCHA assert that »acute bone atrophy» does not occur in syphilitic cases, but PREISER states that their assertion is wrong.) Gonorrheal arthritis is also a recognized cause of the »acute bone atrophy», and DEYCKE-PASCHA mentions lepra nervorum as one of the causes. KIENBÖCK mentions osteocarcinoma — and sarcoma — and arthritis nodosa. (KIMURA says quite contrary to this that arthritis deformans is due to »bone atrophy».) WINTERNITZ, LENK, HIRCHMANN and WACHTEL have found »acute bone atrophy» in connection with frost-bites, REYHER mentions it in BARLOW's disease, and HAIM claims to have found it in rheumatic fever.

Nearly all authors distinguish between the *»acute bone atrophy» originated* by all or nearly all of the *causes mentioned, on the one hand, as distinct from »disuse atrophy», on the other hand*, although most of them mention inactivity as a contributive cause of the origin of the rarefication. (The reason for this peculiar fact seems to be the erroneous idea that inactivity is not able to give rise to a *rapid*

rarefication. Later on (pag. 30) I shall enter more fully into this question.) Thus, all other forms of rarefication do not fall under the heading »acute bone atrophy», according to the classification hitherto employed.

This classification of rarefication holds, however, many perils. A rapid rarefication of bones, brought on by a central cause, resembles also very much the »acute bone atrophy» — as an instance I may here mention NONNÉ's case in which rarefication appeared four weeks after a poliomyelitis acuta. While SUDECK draws a sharp distinction between »bone atrophy» due to a central cause and the »acute bone atrophy» due to a local cause, other authors do not distinguish between rarefication due to central or local causes.

If one reflects carefully upon the above-mentioned causes of the »acute bone atrophy», one must, in my opinion, undoubtedly call in question whether the representation given above is correct or not.

On the whole, it is quite a remarkable fact that so many heterogenous diseases give about the same result — rarefication. Undoubtedly, a general term common to all these conditions may be found; more about this later on (pag. 31).

Many investigators have been at work trying to get an accurate idea of the »acute bone atrophy» and, yet, the problem cannot be considered definitely solved.

As before mentioned, FISCHER wrote an article in 1871 in which he speaks very energetically in favour of the theory of the »trophic nerves». His views seem to have influenced SUDECK and KIENBÖCK who both of them point out that the »acute bone atrophy» is due to disturbances of the trophic nervous elements. Both of them maintain with great vigour that »acute bone atrophy» is not identical with »disuse atrophy».

The proof of SUDECK is as follows:

1) the »disuse atrophy» does not set in as rapidly as the »acute bone atrophy», nor does it reach the same degree of development.

This proof is not of a very great value. SCHIFF and ZAK find in a number of experiments on animals that articular fixation and achillotomy cause an equally rapid and intense rarefication. My own experiments on rabbits show also that rarefication sets in just as rapidly and reaches the same degree of development after having been bandaged with plaster of Paris bandages as after several lesions of various kinds (cf. pag. 24). HAGEMANN's case — described further on — of spontaneous fracture of the femur of a patient who had

been treated with a plaster bandage for luxation of the hip, seems to prove that rarefication from disuse can reach a very considerable development.

2) In many cases there has not been immobilization and, yet, rarefication has set in.

Against this it must be said that, owing to pains or other causes, the patient will himself immobilize to a certain extent the limb concerned, in the diseases mentioned as causes of the »acute bone atrophy».

3) Inactivity is simply a mechanical cause and ought to produce the same changes under the same conditions, but we find — especially after lesions — sometimes great changes and sometimes no changes, in cases where the immobilization has been the same.

SUDECK does apparently not take into sufficient consideration the difference between active and passive movement. But also other factors may come in, especially in connection with lesions can the destruction of the tissues of the soft parts be very different in cases which — clinically and roentgenologically — apparently are alike.

4) The muscular and skin atrophy generally occurring conjunctively. But the acute muscular atrophy is interpreted as being reflex trophoneurotic. (This theory is advanced by PAGET and VULPIAN, advocated by CHARCOT and, in the opinion of SUDECK proved by RAYMOND, DEROCHE and HOFFA.)

Nor on this point is one able to give SUDECK right. The question connected with this matter is treated of in another part of this paper (pag. 33).

5) That the »bone atrophy» may increase also under massage and gymnatic treatment.

This proof is quite insignificant as the action produced by massage and gymnastic treatment is of short duration and of quite a different kind to the natural function.

6) JUL. WOLFF showed that »bone atrophy» is not due to disuse or to epiphyseal injuries, but must be caused by a nervous lesion — according to WOLFF, a trophoneurotic activity.

The proofs of WOLFF against disuse as a cause are, however, identical with those of SUDECK as stated under (1 and 5).

7) Inactivity would not be able to give »spotted atrophy».

I cannot understand why inactivity should not be able to produce it, just as well as other causes. If the changes originate in and develop from a vascular area, then spotted rarefication may arise from all causes. We know that rarefication starts in richly vascular bone parts (epiphyses and marrow) and by and by attacks less

richly vascular parts (compacta), both in cases of rarefication from inactivitity and in the »acute bone atrophy».

NONNE adds two more proofs:

8) He refers to the experiments of SCHIFF — who cut across the motor nerve of the jaw on one side of an animal and obtained a unilateral rarefication.

Against this it must be said that even though the two halves of the jaw be moved together, there is an active movement on the sound side and a passive movement on the injured side.

9) NONNE and CASSIRER point to some cases of hysterical paralysis, in which cases no rarefication set in although the lower extremities were completely paretic.

One must give LEHMANN right when he says that one cannot infer anything from this, as in these cases only those irritations are lacking which reach consciousness, whereas all other irritations may very well be present. (Active movement may perhaps all the same be present in hysterical persons, nota bene when no looker-on is near.)

From my remarks on the proofs brought forward by SUDECK, and others, in order to establish as a fact that the »acute bone atrophy» is not identical with »atrophy from disuse», but is due to the activity of trophoneurotic elements, it will be evident that the whole theory is soaring in mid-air.

MALLY & RICHON think they are justified in connecting the acute muscular atrophy and »bone atrophy» with certain degenerative changes in the anterior horns (cf. the above-mentioned article by FISCHER). And they interpret the rarefication as having originated by a reflex trophoneurotic process.

Others, among whom is BRANDES, suppose the »acute bone atrophy» to be identical with rarefication from inactivity. BRANDES mentions a number of experiments on rabbits as a proof for this assertion. He cut the Achilles tendon on his animals and studied the condition of the os calcis in the roentgen picture. Two weeks — in one single case one week — after the operation he finds a reduced density of the os calcis on the side where the section has been made. The spongiosa is first clearing up and soon afterwards the cortex. Finally, the spongious feature disappears completely and the cortex remains as a paper-thin shell with long fibres. The changes start in the hindmost part of the bone and may be traced to all the tarsal bones and finally also to the tibia. BRANDES lays down the rule that the onset of »bone atrophy» is remarkably quick even in such cases where it is a question only of a partial limitation of function, but

that it is the more rapid and intense the greater the inactivity of the bones is, i. e. the changes are rarefication from inactivity.

The objection may easily be found that a section of the Achilles tendon is undoubtedly a severe lesion which may injure nerves as well as vessels, but BRANDES tries to confute this objection by a picture which shows the vessels to the os calcis not running near the Achilles tendon, and by stating that he has not injured nerves and vessels.

WALTER LEHMANN has for controlling purposes made the following experiment: the Achilles tendon of a rabbit was cut and immediately sutured. After having kept quiet for 2—3 days owing to the painful wound, the rabbit runs about as usual; but there is a reduced density of the os calcis one week after the operation as well as five weeks after it, though then in a lesser degree. From this LEHMANN concludes that there can be no question of inactivity in this case. One must think of lesions of nerves and vessels going to the os calcis.

BRANDES attaches perhaps too little importance to the lesion of nerves and vessels, whereas LEHMANN, on the other hand, pays too little heed to the inactivity. The pains in the wound last perhaps at all events more than a couple of days, and the feeling of weakness in the cut tendon causes surely the rabbit to spare the limb more than LEHMANN is inclined to think. Finally, none of these two authors takes into consideration that infection cannot easily be avoided in operations on rabbits. If the wound is covered up with collodium, the animal itself or the other rabbits will scratch the wound (and if the limb is bandaged, then of course the inactivity will play an important part).

On the basis of 43 cases of rarefication in connection with lesions, ROBERT LENK gives his opinion on this question. He shows how *all joints* in the injured limb, *proximal as well as distal to the lesion, become affected, irrespective of the location of the causal disease.* In diaphyseal fractures the changes seldom develop, or at any rate late — at the place of fracture, more frequently in metaphyseal fractures, but always in epiphyseal fractures and in fractures affecting the joints. He shows further that severe gunshot lesions do not necessarily cause more rarefication than is found in lesions where the skin is left intact. The degree of loss of bone does not always correspond to the degree of muscular atrophy.

The fact that rarefication occurs in a particularly high degree in cases where the inflammation is very severe and where the limb therefore is out of function for a long time, together with the fact that the whole limb becomes affected, indicates that inactivity must be the cause.

The conclusions drawn by LENK seem to me to be too daring, the last-mentioned facts may be comprehended by the theory of reflex nervous activity as well as by theories of the activity of vessels and vasomotor nerves.

To prove his views LENK quotes five cases in which the patients were supplied with plaster bandages adapted for walking, but still did not exhibit any rarefication. This seems rather improbable in view of all the material in hand, thus my own experiments on rabbits also included — though it is quite another thing whether the rarefication was to be seen in the roentgen pictures.

As the rarefication does not appear until three and a half weeks after the lesion, he considers that the immobilization ought not to last for more than three weeks.

Thus LENK as well as BRANDES is of the opinion that the »acute bone atrophy» is identical with rarefication from inactivity.

WALTER LEHMANN has performed a great and elaborate piece of work in order to penetrate in the nature of the rarefication.

His experiences are based upon 40 cases of gunshot lesions of nerves. He considers that the degree of the osseous changes are in the first place due to the innermost structure of the injured nerve, that is to say, to its containing certain centripetal fibres, especially fibres conducting sensations of pain. (K. FLEISCHHAUER also finds the strongest rarefication in cases where the disease is accompanied by pains, and HOFFMANN considers that one should always think of »acute bone atrophy» in intense cases of neuralgia in connection with nerve lesions.)

According to the view of LEHMANN, the development of the process is due to the intensity and duration of the peripheral nervous irritation. He has noticed that the rarefication occurs especially by lesions of the median, tibial and sciatic nerves, and particularly so when the lesions are accompanied by neuralgia; in the next place come lesions of the ulnar nerve. The rarefication is found to be least pronounced by lesions of the radial and peroneal nerves.

The rarefication occurring, as we know, in many diseases that are not accompanied by pains, I find that one cannot very well admit the changes as being due to the moment of pain, except in certain cases, where this moment may be thought to cause a reflex action, e. g. vasocontraction, or to cause inactivity. Moreover, LEHMANN is of the opinion that trophic nerves or sensory nerves with a reversely conducting function are not necessary for explaining the origin of the rarefication. This may be due to a reflex change of the vasotonus, whereby the distribution and concentration of blood and, thus, also the nutrition of the bones is influenced. Matter is

carried to and substance is carried away from the bones. Thus, by circulatory disturbances there arises a deficiency of lime salts. Organic changes of the vessels may also influence the distribution of blood. Inactivity is, according to him, only of any consequence »inasmuch as it may cause a decreased nutrition of the bones».

(My view is exactly the converse of LEHMANN's — inactivity is the essential factor, lesions of vessels and nerves are only of any consequence in so far as they may add to the deficiency in the nutrition of the bones.)

H. HILGENREINER has at his disposal 44 cases of rarefication, all of them results of gunshot lesions. Peculiar circumstances are described in the clinical history of several of these cases. Thus, he finds rarefication of the hand more frequently than of the foot and rarefication of the radius twice as frequently as of the ulna. He sees the explanation of this in the fact that the activity of the hand is greater than that of the foot and the activity of the radius greater than that of the ulna. (Conversely to this, DEYCKE-PASCHA states that the feet are affected by rarefication in a higher degree than the hands in cases of anesthetic lepra.) Now and again he finds the peripheral fragments changed in a remarkably high degree, as compared to the central ones. In a case of multiple fracture, the fragment lying between the two fractures is far more rarefied than the other parts of the bone. Further, he mentions the fact that the head of the humerus shows a reduced density in the roentgen picture, after having been isolated for fracture.

These conditions go to prove that the rarefication is due to nutritive disturbances. For all that, HILGENREINER dare not break off from the SUDECK-KIENBÖCK theory about the trophoneurotic-reflex origin of the rarefication, but considers that these two factors in connection with inactivity cause the rarefication. And, further, he emphasizes the great difference which, with regard to the rarefication, is found in different individuals with lesions of an apparently quite identical kind, treated by the same method. In one case no rarefication was seen, or hardly any — in another case great rarefication. (A parallel to this is seen in the different disposition to callus formation which was first pointed out by ORTH.) From this observation HILGENREINER draws the conclusion that an »individual disposition» to rarefication must exist. (I shall discuss this question further in another place of this paper, pag. 27).

THÖLE rejects categorically the theory of trophic nerves as a condition for rarefication. This is due to the circulation and to the vasomotor nerves.

DAX asserts that lesion of the nutrient artery is of some con-

sequence for the onset of rarefication. He bases his assertion upon observations of patients and experiments on rabbits.

The position of the external opening of the nutrient canal varies, however, very much even in bones of equal length, and the tables set up by DAX of the average position of the nutrient foramen in the different bones, is hardly of any practical importance for the question of rarefication.

FLEISCHHAUER states that the rarefication is due to a »change in the equilibrium», which may arise owing to defective or broken off conduction through both sensory and motor nerves. On the basis of twohundred cases of — mostly — nerve lesions RICH. REZNIČEK tries to prove that the changes in the soft parts and in the bones, following upon the lesions, are due to paralysis of the vascular muscles.

EDINGER is of the opinion that reflexes from the muscles are absent, whereby the effect becomes apparent.

Several authors who are not mentioned here, agree with the view asserted by SUDECK and KIENBÖCK. Some of the opinions expressed are, I think, not very thoroughly considered or not very well founded, as for instance when WEIL states that neuropaths are particularly disposed to rarefication, because in his material of five cases he has one potator and one melancholic, or when COHN says that loss of lime in peripheral segments is due to the lime finding its way to those places where callus is formed. Rarefication is, as we know, not found exclusively in connection with fractures, and it is particularly well pronounced in pseudarthroses.

The points of view which have been referred to above, are the most essential attempts in the literature to explain the nature of the rarefication.

Before I proceed to sum up these opinions and to revise them critically, I shall just mention some experiments that I have made on rabbits in order to throw some light, if possible, upon the problems which the rarefication is so full of.

Altogether I have used twelve young rabbits. They consisted of three different litters of young animals. On eight of them (two litters) the right lower limb was used, and on four the left one.

1) Rarefication from inactivity. Four rabbits had one lower limb bandaged with plaster of Paris — only the femur and crus of one, femur, crus and foot of three. One of the experiments was unsuccessful as an experiment in inactivity (rabbit B), as the animal got an ulceration with inflammation and edema owing to the animal itself or some other animal having bitten its foot. The experiment becomes, thus, a combination experiment in inactivity and infection.

2) Three rabbits had a fracture of crus, which was not bandaged.

3) Two rabbits had a fracture of crus, which was bandaged. In one case a wound appeared.

4) Two rabbits had the Achilles tendon cut.
5) One rabbit had the sciatic nerve cut. In this case infection set in.
After these operations the animals were roentgenphotographed once a week.
The first litter was killed eleven weeks after the operation.
The second litter was killed nine and a half weeks after the operation.
The third litter was killed three weeks after the operation.
The results were as follows:

1) *The experiments in inactivity (plaster of Paris bandage).*

Rabbit A. Slight rarefication 14 days after bandaging, greater rarefication three weeks after bandaging, also some edema. Both of them increase during the following week. The bandages are renewed, the edema disappears. A new bandage has to be applied nine weeks after the first one. The rarefication is fairly unchanged during the last weeks of the life of the animal. Killed eleven weeks after bandaging.

Weight of		Break at a
Os calcis of bandaged side 1.05 gr.		load of 6.75 kg.
» » » sound » 1.18 »		» » 15.0 »
i. e. *about 11 % loss*	and	*more than 50 % decrease of strength.*

Tibia and femur not examined.

Rabbit B. Slight rarefication, some edema fourteen days after bandaging; bandage removed. As the edema persists, no further bandage is applied. A slight inflammation has set in. Some rarefication is steadily seen in the roentgen pictures. Killed nine and a half weeks after bandaging.

Weight of		Break at a
Os calcis of bandaged side 1.22 gr.		load of 11.6 kg.
» » » sound » 1.38 »		» » 14.3 »
i. e. *11.6 % loss*	and	*about 19 % decrease of strength.*
Tibia of bandaged side 9.05 gr.		load of 9.5 kg.
» » sound » 9.30 »		» » 10.1 »
i. e. *2.7 % loss*	and	*6 % decrease in strength.*
Femur of bandaged side 10.88 gr.		load of 10.9 kg.
» » sound » 11.60 »		» » 12.5 »
i. e. *6.2 % loss*	and	*12.8 % decrease in strength.*

Rabbit C. *A distinct rarefication is seen one week after bandaging.* Killed three weeks after bandaging.

Weight of		Break at a
Os calcis of bandaged side 1.06 gr.		load of 9.2 kg.
» » » sound » 1.22 »		» » 14.0 »
i. e. *13.1 % loss*	and	*34.3 % decrease in strength.*
Tibia of bandaged side 7.20 gr.		load of 6 kg.
» » sound » 7.72 »		» » 9 »
i. e. *6.7 % loss*	and	*33.3 % decrease in strength.*

Weight of			Break at a	
Femur of bandaged side	8.16 gr.		load of 7	kg.
» » sound	8.40 »		» » 7.75	»
i. e. *2.9 % loss*		and	*9.7 % decrease in strength.*	

Rabbit D. Only the femur and crus were bandaged. No sign of rarefication is seen in the roentgen pictures. Killed three weeks after bandaging.

Weight of			Break at a	
Os calcis of bandaged side	1.10 gr.		load of 18.5	kg.
» » » sound	» 1.13 »		» » 18.0	»
i. e. *2.6 % loss*		and	*no* decrease of strength to speak of.	
Tibia of bandaged side	7.77 gr.		load of 5.4	kg.
» » sound	» 7.76 »		» » 7.33	»
i. e. *no loss*		and	*26.3 % decrease in strength.*	
Femur of bandaged side	8.97 gr.		load of 6	kg.
» » sound	» 9.33 »		» » 10	»
i. e. *3.9 % loss*		and	*40 % decrease in strength.*	

2) *Fracture experiments.*

Rabbit E. Signs of rarefication are seen *one week after the operation;* pronounced rarefication two weeks after it. Considerable callus formation three weeks after operation. After that, the rarefication is not seen to increase. From the 8—9 week there seems rather to be some new formation of bone trabeculæ and thickening of the cortex, whereas the marrow remains rarefied with scanty trabeculæ. Killed eleven weeks after the operation.

Weigt of			Break at a	
Os calcis of fractured side	1.27 gr.		load of 11.1	kg.
» » » sound	» 1.29 »		» » 16.5	»
i. e. *1.5 % loss*		and	*33 % decrease in strength.*	

Tibia and femur were not examined.

Rabbit F. A slight rarefication and a beginning callus formation is seen two weeks after the lesion. Pronounced rarefication and powerful callus formation three weeks after the operation. Four weeks after the fracture the callus appears to be very solid, but the rarefication seems, if anything, to increase during the following weeks. Killed nine and a half weeks after the lesion.

Weight of			Break at a	
Os calcis of fractured side	0.80 gr.		load of 8.7	kg.
» » » sound	» 0.90 »		» » 9	»
i. e. *11.1 loss*		and	*4.4 % decrease in strength.*	
Tibia of fractured side	7.55 gr.		load of 15	kg.
» » sound	» 5.60 »		» » 17	»
i. e. *increase of weight on account of callus tumor*		and	*11.8 % decrease in strength.*	

Weight of			Break at a
Femur of fractured side	6.10 gr.		load of 11.6 kg.
» » sound	» 6·50 »		» » 15·5 »
i. e. *6.1 % loss*		and	*25.1 % decrease in strength.*

Rabbit G. Doubtful or slight rarefication two weeks after the fracture, distinct rarefication in the third week. Powerful callus three weeks after the operation

Weight of			Break at a
Os calcis of fractured side	0.86 gr.		load of 12.35 kg.
» » » sound	» 0.97 »		» » 16.35 »
i. e. *11.3 % loss*		and	*24.5 % decrease in strength.*
Tibia of fractured side	11.40 gr.		load of less than 11.5 kg.
» » sound	» 6.21 »		» » 15.0 »
i. e. *increase of weight on account of callus*		and	*23.3 % decrease in strength.*
Femur of fractured side	7.0 gr.		load of less than 8.5 kg.
» » sound	» 7.65 »		» » 10.0 »
i. e. *8.5 % loss*		and	*15 % decrease in strength.*

Rabbit H. Slight rarefication two weeks after *the fracture and bandaging.* Five weeks after the operation the bandages had to be renewed. Rarefication increasing. Seven weeks after the lesion the rarefication is very pronounced and remains fairly vigorous until the animal is killed eleven weeks after lesion. (Fig. 9.)

Weight of			Break at a
Os calcis of fractured side	0.80 gr.		load of about 6 kg. (unreliable result)
» » » sound	» 1.05 »		» » 9.4 kg.
i. e. *28.8 % loss*		and	*about 36 % decrease in strength.*

Tibia and femur were not examined.

Rabbit I. Considerable rarefication two weeks after fracture and bandaging. The bandages have to be taken off, as the animal itself or another rabbit has gnawed on its toes and caused a wound. No inflammation is seen. As the animal is very wild and keeps slipping off its bandages, further bandaging is given up. Killed nine and a half weeks after the fracture.

Weight of			Break at a
Os calcis of fractured side	1.3 gr.		load of 9.4 kg.
» » » sound	» 1.5 »		» » 10.8 »
i. e. *13.3 % loss*		and	*13 % decrease in strength.*

Weight of		Break at a
Tibia of fractured side 11.4 gr.		unsuccessful on account of callus
» » sound » 8.35 »		
i. e. *increase of weight on account of callus.*		

Femur of fractured side 10.4 gr.		load of 7.3 kg.
» » sound (left) side 9.7 »		» » 8.3 »
		i. e. *12 % decrease in strength.*

There was a 3 × 2½ cm. large caseous glandular swelling in the angle between femur and tibia of the left side. The remarkable rate of weight is probably due to this circumstance.

3) *Section of the Achilles tendon.*

Rabbit J. Four weeks after the lesion a slight rarefication is noticed which increases a little until the animal is killed nine and a half weeks after the lesion.

Weight of		Break at a
Os calcis of injured side 1.12 gr.		load of between 7.5 and 10.0 kg.
» » » sound » 1.30 »		» » 17.0 »
i. e. *14 % loss*	and	*about 50 % decrease in strenght.*
Tibia of injured side 8.40 gr.		load of 3.55 kg.
» » sound » 8.72 »		» » 6.75 »
i. e. *3.7 % loss*	and	*47.4 % decrease in weight.*
Femur of injured side 10.28 gr.		load of 11.3 kg.
» » sound » 10.52 »		» » 13.5 »
i. e. *2.3 % loss*	and	*16 % decrease in strength.*

Rabbit K. Slight rarefication two weeks after the lesion. Killed three weeks after lesion.

Weight of		Break at a
Os calcis of injured side 0.97 gr.		load of 10.5 kg.
» » » sound » 1.07 »		» » 12.35 »
i. e. *9.3 % loss*	and	*15 % decrease in strength.*
Tibia of injured side 6.63 gr.		load of 4 kg.
» » sound » 7.60 »		» » 9 »
i. e. *12.8 loss*	and	*55.5 % decrease in strength.*
Femur of injured side 7.86 gr.		load of 8.75 kg.
» » sound » 8.22 »		unsuccessful.
i. e. *4.4 % loss*		— — — — — — — —

4) *Section of the sciatic nerve.*

Rabbit L. Inflammation set in very soon. Os calcis became destroyed. When the animal was killed eleven weeks after the operation only some caries-affected remains of the os calcis were found.

Weight of		Break at a
Tibia of operated side 6.40 gr.		
» » sound » 7.15 »		*experiment unsuccessful*
i. e. *10.5 % loss.*		
Femur of operated side 8.5 gr.		load of 3.25 kg.
» » sound » 10.0 »		» » 7.75 »
i. e. *15 % loss*	and	*58 % decrease in strength.*

The weighing was done shortly after death, the bones being carefully scraped and cleansed.

The testing of the strength is carried out in the following manner: The bone is fixed firmly between the chaps of a pair of tongs. Then a lever is measured out to a suitable length, being of course equally long for the right as well as for the left side, but unequally long according to the length and curvature of the pair of bones to be tested. Therefore the weights given are of no absolute, but only a relative, value. A receptacle is suspended at a distance from the tongs equal to the length of the lever. Water is poured into the receptacle with great caution until the bone breaks. The receptacle plus the water is then weighed.

This method is, however, rather unsatisfactory and contains many scources of mistakes: too firm fixation of the bone in the tongs, a slight twist of the bone, alteration of the leverage by a displacement of the receptacle and, finally, too hasty a pouring in of water. Therefore, I consider the figures of the weights to be unsatisfactory in details but, still, I think they give a very useful intimation when taken with reservation. In all experiments where there was no noticeable misadventure, the tests *showed a decrease of strength in the bones of the injured side.* In most of the experiments rarefication could be proved roentgenologically fourteen days after the operation. In two of them — one being an experiment with bandage and one with fracture — rarefication was found one week after the operation.

The most remarkable thing about the figures obtained is that the loss of weight, as far as os calcis is concerned, is practically the same in several of the experiments: inactivity for 11 weeks —11 %; for $9^1/_2$ weeks—11,6 %; fracture $9^1/_2$ weeks—11,1 %; 3 weeks —11,3 %. The experiment with inactivity for 3 weeks gives 13,1 %.

or about the same as fracture plus bandaging, for $9^1/_2$ weeks—13,3 %, and section of the tendon for $9^1/_2$ weeks—14 %. (These numbers tally fairly well with those obtained by NASSE. He found a loss of weight of 13 % on nerve section.)

From these numbers one gets a very definite idea that in these experiments the *inactivity is the only or the all predominant cause of the loss of weight.* That the rabbit E — fracture 11 weeks — shows only 1,5 % loss depends probably upon the fact that the process of healing is almost finished and that the results of the fracture are just disappearing. The new bone elements seem, however, to be far from being so strong as the original ones (33 % decrease of strength).

In the bandage experiment (rabbit D), where the foot is not included in the bandage, the mobility of the foot is reduced a little. In this case the loss of weight of the os calcis is also quite small (2,6 %). In the said case the remarkable decrease in strength, as far as the tibia and the femur are concerned, may perhaps be explained by the high degree of inactivity only in the proximal parts (cf. my theory expounded below about the rarefication as being possibly due to local processes. Pag. 32—33.)

Only in one case — rabbit H, fracture plus bandage — is the loss considerably greater than in any of the other cases, viz. 28,8 %. The reason for this may be that the inactivity has been greater here than in the other experiments, e. g. on account of pains, or it may be due to a firmer fixation of the limb in the bandages, or, possibly, to a combination of these two factors. It seems to me more probable that the cause is a lesion of the vessels and vasomotor nerves, whereby the nutrition of the bones suffers—of course with the inactivity as a contributive cause. It is quite evident, of course, that different fractures cause different degrees of lesions of the soft parts. Further on I shall enter more fully into the different views based upon this fact.

The second case of fracture plus bandage may be characterized as a failure, inasmuch as the bandage remained on only for about two weeks.

Looking at the decrease in strength, one finds altogether very high numbers. *Altogether, the inactivity experiments cause just as great a reduction in strength and rapid loss of substance of the bones as lesions do.*

Several authors have asserted that nerve section does not cause loss of bony substance. My experiment does not give any reliable information on that point. The great loss of weight may, in my opinion, be due to the inactivity caused by the inflammation.

The experiments have shown that one is able to prove a rarefication in the roentgen pictures only when the loss of substance reaches about 10 %—and this only when looking especially for rarefication. *At an ordinary radiological examination a rarefication of 10 % will probably evade discovery.*

Far more accurate is the comparative weighing test which, however, can only be done in experiments on animals. (Still, one must remember that a physiological asymmetry is, no doubt, frequently present, though perhaps as a rule only in a small degree. Furtermore, accidental complications may now and then have some influence on the results — Rabbit I.)

Rarefication can be proved more definitely by the strength test than by the weighing test. The method employed by me, however, is impaired by too great possibilities of erroneous calculations to be used for exact experiments.

The experiments inform us that *not only bones situated peripheral to the bandages or to the lesions, but also bones situated central to these, lose greatly in strength already during the first three weeks after the operation.*

The bones of the affected side were in other respects, as a rule, different to those of the sound side: they were more brittle, broke more easily by pressure with the fingers. They had in most cases a lighter colour than on the sound side. I have not succeeded in finding the »spotted stage» in any roentgen pictures of bones of rabbits. *In the roentgen pictures there has been no distinguishing feature whatever of the rarefication found in the pure inactivity experiments or in the experiments where an operation has been performed.*

As a rule, the loss of substance manifested itself first by a proximal bundle of trabeculæ in the posterior part of the os calcis becoming less distinct. Later one could see that the cortex was getting thinner and that the marrow cavity became clear. The changes were, however, very little pronounced, except in one case, rabbit H.

By the kind help of Mr. C. Steenberg, M. Sc., I have had some of the bones microscopically examined. The bones of the right and the left side gave about the same picture. Numerous osteoclasts were seen lying along the trabeculæ of both the right and the left side, but osteoclasts are always found in great numbers in young animals. Consequently it is difficult to determine whether there are more osteoclasts on the affected side than on the sound side, and I dare not use such a subjective estimation for founding any opinion upon. The histological picture appeared normal in every respect also on the affected side.

It would have been desirable had I been able to bring forth far more numerous and more elaborate experiments on animals. It is no doubt possible in this way to penetrate far deeper towards the solution of this problem than I have been able to do.

If one would collate all the phenomena which have been described in the literature regarding the rarefication and critically examine the numerous explanations that have been given of these, then one would get the following alternatives to judge among:

1) »Disposition» is a necessary condition for rarefication — either expressed as a) »individual» or as b) »neuropathic» disposition (but without any lucid mention of how this is to be interpreted).

2) Rarefication is due to circumstances bound up with the specific motor nerves (not vasomotor nerves).

3) Rarefication is due to circumstances associated with the sensory nerves.

4) GAYET & BONNET intimate cautiously that the sympathetic nervous system is possibly the bearer of the trophic activity.

5) The view maintained by far the most authors that the rarefication is bound up with hypothetical trophic nerve fibres a) either as a functional decline of these or b) (OHLMANN) in such a way that a negative irritation issues from these.

6) The rarefication depends upon the activity of the vasomotor nerves.

7) »Ischæmia», »disturbances of nutrition» etc. causes the rarefication. Such expressions are, as far as I have been able to find in the literature, as a rule employed without any further explanation as to how these are to be understood.

8) The »acute bone atrophy» is in reality the same as »rarefication from inactivity».

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1) »Disposition.» The reason why this idea has been expressed is the freakish origin of the rarefication. In one case rarefication does set in, in another, apparently similar case no rarefication is found. The observations are made especially in cases of lesions. SUDECK admits that he is not able to explain these facts. HILGENREINER's »individual disposition» does not solve the problem, it is only to give the problem a name. Nor does the »neuropathic disposition» of WEIL solve it either. His theory is, moreover, as I have said before, very ill-founded (two nerve patients out of a material of 5 patients)! When one sees rarefication of tibia but not of fibula

(EXNER), or of a fragment lying between two fractures of the forearm, or for instance of a caput humeri fractured and dislocated and, in a far lesser degree, of other bones of the limb, then it can hardly be any question of »disposition». But, on the other hand, one must suppose that local conditions are active in the limb; there are — in my opinion — lesion of vessels and vasomotor nerves. One will then understand that two fractures, exactly similar in appearance roentgenologically and treated in exactly the same manner, may show different degrees of rarefication: the lesion in one case is far greater than in the other.

2) The supposition that the rarefication is due to circumstances associated with the *pure motor nerves* I have seen described by KAPSAMMER as a possibility which he, however, quite coolly disregards when stating his own views. As mentioned on pag. 19, FLEISCHHAUER has also referred to these nerves as possibly being of some consequence for the origin of the rarefication. His theory is, however, as far as I am able to estimate it, rather incomprehensible.

3) *The sensory nerves* may be conceived as playing an important part in the origination of the rarefication in three ways: a) Like CASSIRER, one may suppose that the sensory nerves are associated with a *centrifugal — throphic — function hitherto unknown.* As far as I am able to judge there are no convincing proofs to justify such a supposition, and we may therefore disregard this theory altogether. b) Further, one may suppose that these nerves form the *centripetal part of a reflex arc* which, according to most authors, may be supposed to convey impulses from the injured place to the central nervous system, and from there the impulse that brings on the rarefication is sent back. In my opinion one cannot disregard the possibility that something of this sort may take place through the vasomotor nerves. c) It has been pointed out with regard to the sensory nerves that an irritation of the peripheral parts of a neuron is able to cause vasoconstriction in the regions of other adjacent neurons as a local phenomenon, without the central segment of the neuron being concerned. I have not found this mentioned in the literature as a possible contributive cause of the origination of the rarefication. One can, however, imagine quite well that this circumstance may play a certain rôle in that respect.

Finally, I may mention here the statement made by LEHMANN that »the degree of bone atrophy depends in the first place upon the contents of the centripetal fibres in the injured nerve (in gunshot lesions), and in the next place upon the intensity and duration of the peripheral nervous irritation». Like HOFFMANN, VALENTIN, BLUM, CASSIRER, OPPENHEIM and others he has ascertained the mutual con-

nection between neuralgias and rarefication. It is hardly possible to determine whether this connection is to be explained by the pains causing reflexly a particularly strong vasoconstriction or compelling the patient to keep the limb very much immobilized, or by both of these alternatives together. LEHMANN does apparently not draw these conclusions.

4) That *the sympathetic nervous system* may be of some consequence for the origination of the rarefication is a possibility that cannot be disregarded. But here we enter upon quite unknown problems. Such a connection may be thought to exist in several ways. »Trophic fibres» may be conceived as coming from the sympathetic nervous system just as well as from the central nervous system — and in that case the problem must be treated of under section 5. Sympathetic fibres play an important part in the vascular enervation, and the views that present themselves with regard to the vasomotor nerves, must also be true of the sympathetic nerves. The circumstances associated with the sympathetic nerves are, however, so unknown that I will not venture to put forward any theories on that subject. But I will just mention that PYE-SMITH, SCHIFF and CALLENFELS found a permanent hyperæmia of the affected region after cutting away a piece of the sympathetic nerves. These observations do perhaps indicate that also the sympathetic nerves are of some consequence for the origination of the rarefication.

7) Under this item I have brought together the views which are no doubt correct in all essentials, viz. that *»nutritive disturbances» of the bones* play the principal rôle in the origination of the rarefication. In the literature I have found, however, that these views, with the exception of LEHMANN's, are expressed in a very general way. That »ischæmia» is present in rarefication is, of course, a fact which all who have been studying rarefied bones, have seen; but the views of this kind that I have found expressed in the literature, have not been sufficient to reach a deeper understanding of the nature of the rarefication.

If the views mentioned under 5), 6) and 8) be considered jointly, the following points of view present themselves.

SUDECK ought not to have placed »acute traphoneurotic bone atrophy» in contradistinction to »atrophy from inactivity», for the following reasons: To cause a loss of bone the inactivity must act through the medium of some organ. Which may these organs be? When SUDECK supposes that lesions, inflammations etc. are acting through trophic nerves, then, if he wants to oppose »atrophy from inactivity» to this, he must of course state through which *other*

organs the inactivity may be thought to act. He does not do that, however, nor does any of them who accept his theory.

Or in other words, *inactivity may be opposed to other causes* such as lesion, inflammation etc., but *trophic nerves must be opposed to other organs or means of transmission.*

Is it through the same organs through which lesions and inflammations etc. are active, that inactivity causes rarefication? (Supposing, of course, that the »acute bone atrophy» and the »atrophy from inactivity» are two different categories.)

Almost all authors are of the same opinion that inactivity is a contributive cause — some few say the only cause — of all cases of »acute bone atrophy». All agree further that rarefication sets in when immobilization is sufficiently thorough. The clinical pictures lying obviously so near each other, one may be justified in supposing that both kinds of rarefication (if they really are different) can arise as a *consequence of an activity of the same organs.* Anyhow, no objection has been raised from any quarter to argue against this supposition. Thus, if one accepts the theory that lesions and inflammations etc. are active through trophic nerves, then one has reason to belief that inactivity works through the same organs, i. e. even if one shares SUDECK's *view of the »acute trophoneurotic bone atrophy», one cannot accept his representation of this disease in contradistinction to »atrophy from inactivity»*, except if the theory is founded in quite a different manner to what is now the case.

Is, then, »acute bone atrophy» identical with »atrophy from inactivity»?

Even if, as pointed out above, most of the arguments stated in the literature as proofs of the two categories not being identical are not very convincing (cf. pag. 13), yet many observations go to prove that other factors than inactivity play a rôle as causes of rarefication. I shall only mention the different degrees of rarefication seen in tuberculosis and in other diseases causing just as strong an inactivity as tuberculosis does.

And, further, the tibia may be rarefied and the fibula of the same limb practically unaffected or vice versa, and one fragment may be more rarefied than the others etc. If one considers the atrophy of the skin and muscles to be a manifestation of the same process as the simultaneous rarefication of the bones, then it is, as we know, a fact which already FISCHER stated, that the atrophy of the skin and muscles and the rarefication of the bones may not always reach a relatively equal development. The prognosis of the pure »rarefication from inactivity» is further stated to be far better than the prognosis of many other forms of rarefication.

Although A. SCHIFF & ZAK, on the basis of many experiments on animals, assert that joint-fixation and achillotomy both cause a rarefication equal in regard to its rapid onset and its intensity, and although most of my experiments of rabbits give similar results, this does, however, not constitute sufficient proofs that inactivity is the only existing cause of rarefication. Thus there are, as far as I know, no experimental investigation concerning the bone tuberculosis as a cause of rarefication, and one of my experiments (rabbit H) seems to prove that possibly also other factors than inactivity can be of consequence with regard to the origin of rarefication.

Therefore I think the fact may be established that:

Inactivity is the essential factor in all varieties of the condition hitherto known as the »acute bone atrophy», but that also other factors may be conceived to play a part in the origination and development of many of these conditions.

In other words: SUDECK's observations that other factors than inactivity are of consequence for the origination of rarefication, contain a real kernel of truth, but SUDECK *has over-estimated these factors and, conversely, under-estimated the factor of inactivity. By his list of diseases causing the »acute bone atrophy»* KIENBÖCK *has gone further in the same track, so that later authors have almost completely overlooked the inactivity factor, as soon as there has been another conceivable cause of rarefication.* The consequences have become evident — now and then in a manner anything but expedient, as for instance when OHLMANN recommends to treat patients suffering from »acute bone atrophy» with quiet, basing his recommendation on the supposition that inactivity is a factor of minor importance, whereas the »trophoneurotic» factor is the essential one.

Through what organs does inactivity or the other causes bring on rarefication?

SUDECK & KIENBÖCK were of the opinion that lesions, inflammations etc. were active through some — hypothetical — trophic nerves.

Before assuming that the body has at its disposal some trophic nerve elements in addition to the known sensory and motor nerves and the sympathetic nervous system, there must exist problems that can absolutely not be explained by known anatomical facts.

This is, however, in my opinion not the case as far as rarefication is concerned. In order to substantiate this assertion of mine I shall expound the opinion which I have formed. At the same time I will mention the factors that, along with the factor of inactivity, may, in my opinion, be of some consequence for the development of the rarefication. My view is based upon the anatomical and

physiological facts known to me, but, as there still are many points unsolved in relation to the anatomy and physiology of the bones, the solution of these will perhaps give quite a different conception of the nature of the rarefication than the one set forth here. We know extremely little about the nerves in the bones and, as far as I know, nothing whatever of the distribution of the lymphatics there. The activity of the capillaries in the bones is also practically unknown.

SCHUCKART explains the nature of the rarefication in the following manner: normally one osteoclastic and one osteoblastic activity is taking place in the bones. These two processes equalize each other. In the case of loss of bone the normal loss, which is due to lacunar resorption, is only inadequately made good by a newformation, i. e. *the osteoclastic activity has not increased but the osteoblastic process has either decreased or ceased completely.*

This view seems to be rather convincing.

In what ways may this process be supposed to be started? Hardly by nervous paths. Some anatomists claim to have proved that every cutaneous cell has its own innervation, and some suppose that also all the other cells in the body has their special little nerve fibres. Other believe that, for instance, bone and cartilage cells are not innervated. But, to come to the principal matter, one cannot very well think that osteoblasts and osteoclasts have any innervation, as these cell types are in all essentials more like leucocytes (phagocytes); and one can hardly conceive these cells being linked by nerve fibres. *One must suppose that these cells are influenced by chemical means,* i. e. probably *by matters from the capillaries or, vice versa, by the failure of the matters* normally supplied. (As a kind of parallel one may in this connection think about the influence which the internal secretion has on the bone tissue, for instance in acromegaly, or about the locally produced, unknown matters that cause a breaking down of the cartilage during the growth of bones.)

It is, as we know, a fact that the capillaries play an important part in all questions concerning the nutrition of tissues. And it is extremely probable that the capillaries also play a part in the process of rarefication. The phenomenon described above, that edema is very often ascertained in connection with rarefication, favours this view. But then I shall not enter upon theories at this point.

If we pass on to the case of the large vessels, especially of the arteries, then the following points must be taken into consideration; firstly, the possible rôle played by the sensory nerves in the origination of the rarefication (mentioned under 3), that *vasoconstriction may take place by the conveying of an irritation through the peripheral*

net of sensory nerves without any central co-operation (BAYLISS, BRUCE, A. KROGH and others), and secondly, the fact *that when a muscle is active, a great amount of blood flows through it independent of the action of the muscle nerve*, even if this is degenerated. Thus, the reverse case is also a fact, viz. that *on account of purely local conditions muscular inactivity causes a lesser circulation of blood* than when the organ is active in a normal manner.

There is also the possibility *that the amount of blood in the organ is lessened owing to reflex vasoconstriction.* That the vasomotor nerves are extremely susceptible to reflex influences is a fact that is proved by our daily experiences.

If we look especially at the behaviour of the arteries in fractures, nerve lesions and inflammations, the following considerations present themselves:

Arteries may be injured when lesions occur, and these arteries will be blocked up by thrombosis. If the lesion is sufficiently extensive, an effective collateral circulation will hardly be re-established so easily (fract. colli hum. anat., fract. colli fem., fig. 10, the middle fragment in HILGENREINER's multiple fracture etc.). In such cases we find, as a rule, a particularly strong rarefication. In cases of less interfering lesions, the same will happen, though in a lesser degree.

Concerning nervous lesions one may suppose that in less severe cases a state of great irritation of a relatively lasting character sets in, thereby causing a vasoconstriction with concomitant decreased circulation through the peripheral parts of the limb. Section of nerves produces a dilatation of the arteries (which has been observed by KAPSAMMER, BERVOETS, LAPINSKY, FRÄNKEL and others). This vasodilation is, moreover, manifested by the above-mentioned hyperæmia of the marrow and periosteum (SCHIFF) and by a rise of temperature in the limb during the first 4 to 8 days after the operation (KAPSAMMER and others). And, further, a »thickening» and »infiltration» etc. of the vascular wall has been ascertained. (A. FRÄNKEL, CZYHLARZ & HELBING and others.) It may easily be supposed that such changes in the arterial wall may continue down into the capillaries and thus make them narrower, or injure the cells of the capillary walls. A lasting arterial dilatation in connection with the increased blood pressure and the simultaneous slow circulation, which has been mentioned (LAPINSKY), may perhaps also be injurious to the capillaries, in such a manner that their function becomes abnormal.

Anything that takes place on section of a nerve may also take place in case of other complete destructions of nerves, whatever causes these destructions be due to.

In various inflammations one finds often thrombosis of vessels. These cloggings may result in the supply of blood, for instance, to the bones, becoming less than normal. In cases of inflammation one must also remember the peculiar affinity to nerve elements which many bacterial poisons have (diphtheria, syphilis, lepra — the two last ones are, moreover, mentioned among the causes of the »acute bone atrophy»; as to syphilis, its affinity to the arterial walls may also be mentioned here). The fact that some infections cause a stronger rarefication than others may perhaps be explained by the circumstance that the affinity to the nerves is unequally great for different bacterial poisons, in connection with the also unequally great tendency to thrombosis that is found in different inflammations.

When a nerve is injured, the peripheral portion is completely destroyed, and the axis cylinder of the central portion, together with its collaterals, is also degenerated, at least a short way up. CASSIRER says: »Section of the axiscylinder causes a quickly appearing change in the structure of the corresponding nerve cell in the anterior horn». If this be the case, then perhaps, it may also be conducive to the rarefication attacking the parts of the limb situated proximal to the causal factor. This last phenomenon can, moreover, be explained in various other ways: partly as a result of the above-mentioned lesser supply of blood due to inactivity, partly as an outcome of the above-mentioned local transmission of nervous impulses, together with the vasoconstriction due to this, partly on account of the vasoconstriction through the usual reflex arc and, finally, as a consequence of the vasodilation due to failing reflex impulses.

The notions are very hazy on this point. Closely related to these questions are the problems concerning the forms of rarefication due to causes from the central nervous system, wich problems are beyond the scope of the subject treated of in this article.

From what has been said it is evident, I think, that inactivity is the essentail of the origination of rarefication. *Inactivity must be supposed to work through the circulation. Factors acting in the same direction as inactivity, viz. lowering the function of the circulation, may be supposed to have an increasing effect on the result of the inactivity: rarefication* (and atrophy of the soft parts). On the basis of this hypothesis one is able to understand how it is possible that causes, so widely different as tuberculosis and lesions (which, in my opinion, may undoubtedly increase a rarefiction due to inactivity), can produce exactly the same result. These two factors are acting on the bones, although perhaps in different ways, through the medium of the same organs, vessels and vasomotor nerves, and therefore *the changes seen in the roentgen pictures of the bone became identical.*

That the rarefication is bound up with the function of the vessels I consider to be supported by the fact that the changes are first manifested in the most vascular portions of the bones, i. e. the epiphyses and the trabeculæ in the marrow. And here the process develops most strongly.

The »spotted atrophy» may be conceived as due to the changes starting in the regions of small vessels and then spreading further by degrees. The theory of the »trophic nerves» seems hardly likely to explain the origin of the »spotted stage».

All the facts stated by SUDECK and others seem to be explainable by the above-mentioned theory.

Several experiments on animals which have been used in support of the theory about the »trophic nerves», may likewise be understood by the above theory. This applies, for instance, to the experiments by SCHIFF, which have been described above.

If, for controlling purposes, one investigates as to whether kindred diseases can help us towards a proper understanding of the nature of the rarefication, it is only natural to consider other forms of »bone atrophy». In the arteriosclerotic form one finds that changes in the vessel-walls are the most natural explanation of the loss of bone, of course, even if a reduced activity plays an important part also in that case. »Pressure atrophy» is, in my opinion, extremely closely allied to the »acute bone atrophy». The cause is a lesion, the effect is a bad nutrition owing to an arrested circulation, which in its turn is due to a compression of vessels, possibly in connection with irritation or destruction of nerve elements. These processes are then parallel to those which are supposed to be the cause of rarefication from lesions, inflammations etc.

In the literature there are very few opinions of any consequence which contradict the theory that has been advanced above. FISCHER'S above-mentioned objections to the interpretation of the condition of the vasomotor nerves as being of any importance for the origination of the rarefication, is of course of no value whatever. DEYCKE-PASCHA states that rarefication is not seen in mb. cordis, arteriosclerosis (which, of course, is absurd), mummification or gangrene (in the two last diseases it is a question of *interrupted* circulation — thus, a cessation of osteoclastic as well as osteoblastic activity). On these grounds he thinks he may exclude the possibility that the cause of the rarefication is to be sought for in the organs of circulation.

On the other hand there are, as mentioned before, fairly many opinions expressed in the literature tending in the same direction as the theory that I have advanced above, even if nobody — at

least as far as I have found — has explained in detail the nature of the process in the same manner.

While, thus, the generally established opinion purports that the »acute bone atrophy» in connection with lesions, inflammations, tumours etc. is one form and the »inactivity atrophy» quite another form, I think we might lay down as an incontrovertible fact that both forms are essentially one and the same process. The changes caused by inactivity can be increased by all the factors that have an injurious effect upon vessels and vasomotor nerves.

(When I employ expressions such as »lesion rarefication», »inflammatory rarefication» etc., these expressions signify consequently »rarefication caused by the inactivity due to lesion, inflammation etc. and possibly increased by special factors dependent on the nature of the disease».)

The diagnosis.

It is generally necessary to make the diagnosis, rarefication by local causes, by comparing the density of the bone shadows of both sides. (It goes without saying that the extremities must be placed in symmetrical positions, that the tube must have the same degree of hardness at both exposures, that the exposures must be of the same duration etc. Technical faults in photographing may cause one to overlook or overestimate the rarefication. But then I shall not enter upon the technical side of the matter which every roentgenologist is familiar with.)

Clinically, muscle atrophy, hard edema, cyanosis etc. will afford valuable hints in cases where the anamnesis may possibly cause one to suspect the presence of rarefication. Pains are not generally found in connection with rarefication.

The question of simulation is easily settled by a roentgen examination.

The spotted stage of the rarefication may be mistaken for other complaints, and the difficulties are increased by the fact that practically all bone diseases are able to cause inactivity and, thus, also rarefication. Multiple bone cysts (RECKLINGHAUSEN), myelomatosis (KAHLER) and bone tophi in arthritis urica show well-defined clear areas in the roentgen picture and affect as a rule the right as well as the left extremity. At a high development of these diseases, at a point where the areas of reduced density coalesce, one finds as a rule a destruction of bone segments in arthritis urica, or one may find normal bone tissue here and there between the coalescing areas. Nor does the size and distribution of the spots resemble what is seen in the case of rarefication. This is, as we know, most pronounced in the epiphyses. Moreover, the clinical history will as a rule afford sufficient information to prevent every mistake. In cases

of long duration the picture of a single segment of the extremity may of course show changes, that are due to inactivity rarefication. A comparison with photographs of other segments will give the necessary information.

Cancer metastases in bones may resemble spotted rarefication to some extent (Fig. 11). Cancer metastases of this kind are found especially in cancer mammae or cancer prostatae. They seem to be disposed to become localized more particularly in the central portions of the skeleton — the spinal column and the pelvis — than in the peripheral portions of the extremities, where the spotted rarefication is to be found almost exclusively. Nor are the cancer metastases seen to be concentrated particularly in the locality of the epiphyses, as the rarefication is. Osteosclerotic patches are not unfrequently found in cases of cancer metastases. Besides, the bone metastases are of a somewhat different character to the rarefication: numerous, about equally large areas with a not very distinct rarefication — or perhaps entirely without it — in the surrounding bone tissue which often has a peculiar »cotton-wool» appearance, in contradistinction to the unequally large, diffuse areas and »blots» in an obliterated, rarefied surrounding bone tissue.

»Ostitis rareficans» is a term by which different authors mean different things: (LISSAUER has, for instance, described a case of traumatic bone disease as »ostitis rareficans», while SUDECK describes a similar case as »acute bone atrophy». GOTTSCHALCK mentions ostitis rareficans» and »caries sicca» without emphasizing the difference between these two terms, and PHEMISTER speaks of »rarefying osteitis» as a zone lying at a variable distance from infectious destruction of bone). Without entering further upon the definition of »rarefying osteitis», I think one might be justified in asserting that in cases where there are signs of periosteal irritation, destruction of the contours and focal clearing-up, it may undoubtedly be a question of osteitis in one form or another, whereas such phenomena are not seen in the bone segments that are *solely* rarefied. In practice, however, one comes now and then across areas of reduced density in cases of rarefication, which areas can resemble foci (fig. 12. It is a case of rarefication after an ordinary (common) fracture of the radius treated with plaster bandage. Clinically, there is nothing whatever to indicate inflammation. One notices a large, focus-like area of reduced density in the styloid process of the radius.) Another point is that an inflammation of bones is accompanied by rarefication. Fig. 13 shows the features which have been described as characteristic of osteitis, in combination with a high rarefication in a foot that was operated after a fracture.

The greatest difficulty met with in the department of diagnostics, as far as the rarefication is concerned, is the fact that tuberculosis of soft parts and of bones is nearly always followed by a particularly great rarefication in the surrounding osseus tissue. The difficulties are increased by the tuberculosis being very often preceeded by a trauma (BRUNS states that in tuberculosis of bone a trauma can be proved in 14 % of the anamneses, KÖNIG in 20 % and MIKULICZ in 29 % — this refers to tuberculosis in definite regions). A careful search for foci and indistinct or »gnawed-off» contours will, as a positive finding, give the diagnosis of tuberculosis of bone, whereas a negative finding does not preclude the possibility that tuberculosis of bone or soft parts is the cause of the changes. To state definitely where the tuberculosis ceases in a tuberculous bone and where the tissue is thinned only by rarefication, may be an unsolvable difficulty, especially in the »spotted stage of rarefication», Fig. 2.

When the second stage with its sharply-defined bone trabeculæ is reached, »woolly» diffuse portions of a bone with an otherwise distinct structure should be interpreted as tuberculous segments, Fig. 3. Repeated photographs, especially when taken at intervals of one or more weeks, will further establish the extension of the process.

A pronounced rarefication will in doubtful cases favour a diagnosis of tuberculosis, as this disease is particularly predisposing to rarefication.

As to the diagnosis rarefication, it should be borne in mind that *this is a symptom* caused by a disease or a condition, and that the point is to trace this casual factor in cases where it is not clinically indicated beforehand.

Wherever it is a question of rarefication, one must always take roentgenograms of both sides and remember that the rarefication is not necessarily localized in an extremity, but may be due to arteriosclerosis or some other universal or central cause. Changes which are due to such a non-local cause cannot, as a rule, be distinguished in the single picture from a rarefication due to a local cause.

For the sake of completeness some differential-diagnostic characteristics of certain bone diseases will be mentioned below.

Osteomalacia has its own special character with thickenings and deformities of the bones, and the localization of the disease which is not confined to any certain region, as well as its anamnesis and clinical features, will prevent it from being confused with rarefication. One must remember, however, that osteomalacia has originated in many cases in connection with a trauma.

Rachitis occurs, as we know, at certain periods of life, it is relatively symmetrical, the anamnesis and the clinical course give such a typical picture that the roentgenologically ascertainable changes, by which this disease is distinguished from the rarefication, hardly need to be mentioned.

»Osteopsathyrosis» is employed partly in the sense of fragility of the bones and partly to characterize a certain disease. This is a very rare disease which is characterized by the great tendency of the bones to fracture (thus, BLANCHARD mentions the case of a 12 years old girl who had had 41 fractures since her second year!). This disease is hereditary, begins at the age of 1—3 years and is not localized to one single extremity or region.

Clinically, painful flat-foot can give the same picture as rarefication. The anamnesis and the roentgen picture will probably supply the necessary differential-diagnostic characteristics in most cases.

Prognosis.

The importance of recognizing the rarefication and its prognosis is evident in all essentials from what has ben mentioned above.

A slight or moderate rarefication is of no consequence in most cases.

But if the rarefication reaches a high stage of development, it becomes a complication to which one must attach great importance as being a bad prognostic with regard to the time it will take to obtain a recovery as well as with regard to the chance of an eventual restitutio ad integrum.

Now and then one finds, of course, that the rarefication improves rapidly, but this is not at all a general rule. The rarefication enters very often into a chronic stage.

It seems to be a fact that one gets more frequently a satisfactory improvment of the rarefication than of the accompanying changes of soft parts. Many diseases of joints are particularly seen to result in a bad function, especially springy stiffness — probably owing to a shortening of ligaments and capsule. It has, however, also been stated that the changes in the soft parts may disappear without the bony changes disappearing.

The pains that may accompany the rarefication, especially in the lower extremities, disappear as a rule in time, but a certain disability — particularly in the feet — may remain for years. The feeling of lack of strength in the extremity may also persist for a very long time. This fact is bound up with the tendency of the muscles to remain atrophic for years. HAENELL found a considerably muscular atrophy in eleven out of his forty cases of fractures — later on the atrophy disappeared in five of the cases. CHAPUT got

similar results. One to five years after the fracture had occurred SVEND HANSEN found muscular atrophy in 74 out of 245 cases of fractures of the ankle-joint (the atrophy being 2 cm. or more in 36 of the cases). In 30 cases he found edema of the leg and foot five years after the fracture.

Inactivity rarefication is said to have a far better prognosis than the lesion rarefication, but I have nowhere found a report of any investigation that proves this assertion. In such cases where there is no organic destruction of vessels and vasomotor nerves, it seems probable that a reparation may take place more easily than in cases where there is such a destruction.

With regard to the formation of callus, LENK thinks he can prove that rarefication cannot be considered to be a cause of a slow callus formation, as he has had cases of high rarefication with plenty of callus. BIER pronounces an opinion to the same purport as LENK, while HILGENREINER, CORSO and OHLMANN are of the opinion that rarefication can cause an insufficient callus formation and slow consolidation. I think I have found that *the tendency to heal is often slow in cases of great rarefication*, but I think that the cause of the slow healing and of the rarefication is the same in both cases, thus, that the rarefication is a symptom of bad circulation and that this, on the other hand, may involve that the disposition to heal is not so good.

The tendency to pseudarthrosis in cases of rarefication which, for instance, HILGENREINER describes, must, I think, be understood in the same manner.

Rarefication has further been blamed as a cause of bad results of operations. It is, of course, very probable that, *in cases where the tendency to heal is slow owing to the nutrition of bones being bad beforehand, every further lesion of vessels and nerves must have a most unfortunate effect.*

One would expect the decrease in strenght, as far as rarefied bones are concerned, to result frequently in deformities or fractures. It is a remarkable fact how seldom such a thing is described in connection with rarefication from lesions or inflammations. The reason for this is possibly that sufficient attention has not been paid to causative relation on this point.

Deformities and fractures are, as we know, common occurrences in senile bones. SCHUCKARDT mentions both bendings and infractions of bones after acute poliomyelitis. Fragility of the bones plays an important role in syringomyelia (TEDESCO) and what we see in tabes dorsalis also points to the rarefication of bones being of great consequence in causing deformities as well as fractures.

Deformities of rarefied bones are mentioned by OBERST, KÖNIG, SCHEDE & STAHR, SCHARFF and OHLMANN and others in osteomyelitis. HILGENREINER, too, mentions 2 cases of deformities, and DEYCKE-PASCHA opines that deformities — especially of the lower extremities — in connection with lepra nerv. are due to the rarefication accompanying that disease.

In spite of the considerable decrease in strength caused by rarefication (cf. my experiments on rabbits), one hears very seldom of fractures in bone tuberculosis or of refractures after common fractures. The reason for this must be that the diseased bones are spared involuntarily. On looking through 700—800 journals on fractures in »Arbejderforsikringsraadet» (Workman's Insurance Commission) I did not find a single case where a refracture could be said to be due to a rarefication after a primary fracture.

But, still, there are some cases in the literature that prove that fractures in connection with inactivity rarefication, inflammation rarefication and lesion rarefication are not so very rare after all. SABRAZES & MARTY mention a patient 27 years of age who at the age of twelve had a luxation of his right shoulder. During the following years the patient had a fracture of the right upper arm twice and of the right clavicle once. Muscular atrophy set in as a result of the first trauma and became very pronounced. Thickenings of the skin and a hypersensibility to cold and heat were concomitant results. The authors suppose that the first trauma has injured the brachial plexus.

RICH. HAGEMANN, EHRINGHAUS and FROMME mention altogether 11 cases of a special type of fracture of the thigh, which is due to rarefication. All cases occurred in connection with plaster bandages. HAGEMANN's only case was a patient suffering from luxation of the hip (thus a pure inactivity rarefication). In nine cases the patients were treated with plaster bandages for tuberculous coxitis. In the case described by FROMME the patient was suffering from tuberculosis of the knee. The first-mentioned patient got two spontaneous fractures of the diseased femur after the plaster bandage was taken of — the first time during the massage and the second time when trying to walk. All the others got their fractures in a similar manner, only through a very trifling cause. All the fractures were situated just above the condyles of the femur. HAGEMANN explains in detail the technic of the fracture. EHRINGHAUS believes that it is a question of tuberculosis of the bone, but this supposition is disproved by the case of luxation. FROMME's patient got his fracture while the leg was lying in its plaster bandage — and it healed before the bandage was removed. PAUL BRUNS describes a similar case in 1886. To

this number can I add one case which Dr. AAGE KOCK has kindly communicated to me: About 1908, a boy of eight was treated for coxitis with plaster bandage. When the bandage was removed, he got a spontaneous fracture of the femur.

Without entering further into the question HILGENREINER mentions that he knows several cases of fractures due to fragility caused by rarefication, and KIENBÖCK states that a new fracture can occur when one is testing the strength of the healed fracture after the termination of the treatment.

The examples given should be sufficient to prove the importance of rarefication.

Finally, I shall mention the prognostic importance of the rarefication with regard to growing individuals. The rarefication in such individual is, as I have mentioned above, as a rule accompanied by a concentric atrophy of the bones, i. e. their growth is retarded.

Prophylaxis and Therapeutics.

Inactivity of extremities should as far as possible be avoided. When it is necessary to immobilize an extremity, the degree of immolization should be as small and the duration as short as possible.

Further, the causal therapy must be applied in every case in such a manner that *the course of desease is shortened as much as possible* and *vessels and nerves injured as little as possible.*

Finally, *as early as possible one should employ massage, passive and active movements, baths and electrotherapy.*

When these principles are being followed in the therapy of fractures, they mean, that repositions must be performed in such a manner that the fragments are placed in the most favourable positions, *yet without the soft parts being further injured during the reposition.*

Operative treatment of fractures must, as far as possible, be avoided. Still, if operations become necessary, as *few vessels and nerves as possible* should be cut across; thus *the incision should be as small as possible, vessels and nerves should be pushed aside rather than be cut, the periosteum should not be removed if it can be avoided, etc.*

As regards bandaging, a roll-pad dressing is to be preferred to a plaster bandage, a removable bandage to an unremovable, a walking-bandage or an extension device to a firm, stiff and close-fitting bandage—of course with due allowance for the circumstances prevailing in each separate case. *The dressing should not be larger than is strictly necessary.* If the injured placed cannot itself stand early

massage, then one ought to start massaging the sound parts of the extremity. One need not wait to give movements until wounds (if they are present) are completely epithelialized. OHLMANN recommends treatment with quiet in cases of rarefication, on the theoretical supposition that inactivity is an immaterial factor in rarefication. From what has been said it is probably evident that his view is wrong.

SOMMER recommends to have electrodes introduced into the bandages so as to be able to apply electricity to the muscles while the bandages are still on. SUDECK recommends HELFERICH's treatment with stasis. BLECHER, THIEM, CARL WESSEL and others are using BIER's stasis. (I suppose this therapy is founded on the observations made by DUMREICHER and others that venous stasis causes an increase in bone tissue).

Every therapy having a healing effect on the disease from which the rarefication springs, will be well adapted to combat the rarefication. That light-baths must be considered particularly appropriate in cases of rarefication in connection with tuberculosis, is a fact. The opinions advanced with regard to operations of fractures apply, of course, also to the operative treatment of inflammatory diseases.

When patients suffering from a high degree of rarefication in the lower extremities get up, one must be extremely careful in letting them try to walk during the first time. Massage, too, must be given with care to begin with, and every manipulation, e. g. testing the strength of a united fracture, must be done with great caution.

Internal treatment with lime seems to be theoretically unfounded. I do not know what results have been obtained by this method of treatment.

The rarefication is of great interest from an *insurance point of view*, yet only in such cases where it present in a considerable degree and is of long duration.

From what has already been said it is evident that the rarefication means a drawback to the patients in several ways:

Subjectively—because this state is accompanied by lack of strength, often by pains and often by stiffness in the joints. This stiffness is especially frequent in the wrist and may involve a high degree of invalidity. The pains are mostly localized to the lower extremities (load pains) and may consequently greatly diminish the working-power of the patient.

Objectively—because of the bad prognosis of the case when the

rarefication is highly pronounced. As rarefication is a symptom of another disease or morbid condition, a claim for damages should not be rejected in such cases where there is a considerable rarefication, even if no further clinical phenomena can be ascertained. It is perhaps best to settle such a question temporarily with a chance of reconsidering it (the possibility of tuberculosis), or else the damages ought to be paid.

A lesion accompanied by strong rarefication ought to have higher damages than a similar lesion with no or with only a small rarefication, but of course with due consideration to the subjective phenomena.

I order to study more closely the practical consequences of lesion rarefication I have examined some material from the Arbejderforsikringsraad (Workman's Insurance Commission) in Copenhagen. I am of opinion that all fractures affecting the joints give a very variegated picture which is often difficult to understand owing to coexistent changes in cartilage, capsule etc., and that as a rule it is impossible to determine which subjective and objective symptoms are due to the soft parts and which to the rarefication. For that reason I have only collected diaphyseal and metaphyseal fractures, which do not affect the joints. And, further, I have only included fractures of the long tubular bones. These considerations are, however, only applicable to the first group of cases. Altogether I have examined between 700 and 800 cases, of which only a part had been photographed with the X-rays. Only 44 of these cases seemed to be of any consequence for the matter under consideration.

I have divided my material into three groups.

1) I have examined a number of diaphyseal fractures (as mentioned).
2) » » » the cases collected in the card-library under the heading »Halisteresis».
3) » » » the cases collected in the card-library under the heading »Refractures».

The last group, which contained rather few cases, I looked through in the hope of finding some cases where a refracture had occurred as a result of the particularly strong rarefication after the first fracture. I did not manage to find any such case.

In estimating this material one must, however, remember that nearly all the roentgenograms were taken about 1 year after the lesion. Thus, »acute» is not really a suitable name for the loss of bone that was found to exist. As a matter of fact, the material that I looked through gave me very little information of interest. While the changes in the rarefied bones as a rule showed a diffuse

reduced density in the roentgen picture that is characteristic of the second stage or the chronic form of rarefication, I found now and again spotted rarefication which, of course, is a rather unique thing to find such a long time after the fracture. In one case this spotted stage was found in a patient suffering from arteriosclerosis, and in another case in which a patient had got inflammation of a knee after a contusion whithout fracture. In this case the spotted picture may perhaps be due to the inflammation of the knee which was of a later date than the lesion. In one or two cases of spotted rarefication no reason could be ascertained why the rarefication was spotted, either clinically—by means of the journal—or by any other means.

Twice I found a considerably stronger rarefication of the fibula than of the tibia, and once I found an extraordinarily heavy loss of bone in the uppermost part of the os calcis and only a moderately heavy loss in the leg and foot.

Like so many other investigators who have occupied themselves with these questions I found nearly always a distinct muscular loss when rarefication was present, but not in a regular proportion. There was often a heavy muscular loss in connection with a slight rarefication of the bones, or vice versa. Very often the card mentions hard edema, especially in such cases where plaster bandages have been used.

Pains on walking are reported in most cases where the leg has been affected by the rarefication. Now and then the patients did not complain of pains but only of fatigue.

In one case of rarefication there is only contusion and no fracture of the left leg. The patient has been in bed with plaster bandages for half a year and has had no massage. When he returns to be re-examined after a year, he walks badly, has hard edema in leg and foot, and the mobility of the foot has almost disappeared. The muscular loss of the calf is 2 cm. and that of the thigh 4 cm. In a case of this kind one might think of tuberculosis, but as the matter does not seem to have been brought up for reconsideration one year after having been settled, it is perhaps only a case of highly pronounced inactivity rarefication.

I found one case of contusion without fracture and several cases of fractures healed in good position, all having considerable rarefication. And, vice versa, I found several fractures that were extremely badly united but showed almost no rarefication.

The material is very varying. Some cases are treated with plaster bandages, others with early massage — the patients coming, of course from different doctors and different hospitals. The informa-

tions as to the nature of the treatment are in most cases very uninstructive, for instance when the heading »treatment» is exchanged for the information »bandage» without any mentioning being made of its nature or of the time it has been applied.

Damages have been paid all through to an amount equalling a degree of invalidity of from 40 % down to 8 %. The estimations seem to have been satisfactorily done in every case.

With regard to the question »simulation or rarefication» the roentgen picture will, of cource, help us to overcome the difficulties. Pains from pressure, caused by callus, alcoholic neuritis, tabetic pains etc., are mistaken for pains from loads in cases of rarefication.

At an inadequate roentgen examination rarefied bones can be interpreted as normal. In photographs taken from the affected side only, an arteriosclerotic loss of bone may, for instance, be interpreted as rarefication due to lesion. Thus, in all cases where an insurance claim is to be settled, it is absolutely essential to make use only of roentgen pictures that are taken with a perfect technic, and control pictures of the sound extremity should always be demanded for comparison.

I am under a great obligation to Professor ELLERMANN, Professor A. KROGH, Dr. H. J. PANNER, Dr. FOLKMAR, Mr. C. STEENBERG Sc. M., and Dr. F. DJÖRUP for all valuable hints they have given and for the kind help they have extended in many ways. I am also greatly indebted to Board of the Workman's Insurance Commission for the permission to make use of the material obtainable in the journals of the Commission.

SUMMARY

The clinical picture which was first described by SUDECK as the *»acute trophoneurotic bone atrophy»* and then by KIENBÖCK as the *»acute bone atrophy»* and which is presented as a *contrast to the atrophy* by inactivity, is inaccurate and misleading — both from a theoretical point of view and with regard to the term employed.

1) The following considerations present themselves when the matter is looked at *from a theoretical point of view:*

a) »Trophic» nerves are (hypothetical) *organs*, inactivity is a *cause of a pathological state. These two ideas can therefore not be placed in direct opposition to one another.* In contradistinction to a loss of bone by the way of »trophic» nerves one must set a loss of bone via other *organs.* Against loss by inactivity one must oppose a loss

due to other *causes*. There is no ground for the supposition that inactivity exercises an influence through other organs than those by which the diseases and conditions that are pretended to be the cause of the »acute bone atrophy» are active.

b) There seems to be *no reason to suppose that »trophic» nerves are of any importance* in the condition known as »acute bone atrophy».

c) *The chief cause of the appearance of the »acute bone atrophy» is undoubtedly inactivity. Still, lesions of vessels and nerves — both of a toxic and of a traumatic kind — can probably be conducive to the development of loss of bone tissue.*

2) With regard to the term »acute bone atrophy» the following considerations are applicable:

a) *The changes are not »acute»* according to the general definition of this word.

b) The »acute bone atrophy» is a term formed as a parallel to the name of the simultaneously appearing changes in muscles — »muscle atrophy». But while »muscle atrophy» means *diminution of size*, »bone atrophy» is a *change in substance*.

The change in muscle corresponding to this change in substance is called *»muscle degeneration»* (fatty degeneration).

In order to get a distinct nomenclature I therefore propose that »bone atrophy» should denote a *diminution in the size of the bones* (concentric atrophy) whereas *degenerative changes in the substance of the bones* (eccentric atrophy) should be called »bone degeneration» (or »lipomacia»). The term »rarefication» may be employed to express bone degeneration in a roentgenological meaning.

After having described the clinical picture, the author enters upon a critical examination of the many diverging views regarding about the development, causes and explanations of the disease. He considers himself justified — on the basis of his experiments on animals — to establish as a fact that a diminution of bones *always* takes place in connection with severe lesions of extremities, of whatever kind these may be, but that this diminution can not be recognized in the roentgen picture when present only to a small extent. A rarefication of about 10 % is, as a rule, roentgenologically ascertainable in the bones of rabbits.

The spotted stage can probably only be observed in high degrees of rarefication. As a rule this stage appears fairly soon after the causal factor and disappears in the course of a few weeks or months, but the author has found it one year after the lesions had taken place.

The author's experiments on animals show *in a number of cases practically the same rapid origination and strong development of rarefication in connection with diverse lesions* as in *pure inactivity experiments* (plaster-bandaging) — all investigations have been carried out on the lower extremites of rabbits. *The bones in the affected extremity showed a constant decrease in strength.*

Rarefication from inactivity and from lesions gives exactly the same roentgenological picture.

The theories that have previously been advanced in the literature and which pretend to explain the nature of the »acute bone atrophy», are divided into 8 groups and critically examined. The author then expresses his own opinion which he bases partly on the literature, partly on his own observations and partly on his experiments on rabbits. And further it is founded

on certain physiological observations and theories and culminates in the hypothesis that *local processes associated with the vessels and their nerves can explain the problems connected with this matter. It is further supposed that causes from the central nervous system or the sympathetic nervous system may be of some consequence.* This influence is thought to be bound with in the *vasomotor nerves.*

Under the heading »diagnosis» the author emphasizes especially the relation between rarefication and tuberculosis.

The author is of the opinion that the rarefication may be of a considerable prognostic value, and substantiates this view more closely.

Some prophylactic and therapeutic conclusions are drawn, the chief object of which is to urge to caution in reposition of fractures, to point out the necessity of sparing fractured bones as much as possible in operations, and to recommend the massage, active and passive exercises to be given as soon as possible during the clinical course. The main point is, however, to avoid immobilization of extremities as far as possible, or at all events to reduce the immobilization to the shortest possible duration and to the smallest possible degree.

Finally, the author sets forth in a short section some views regarding the importance of the rarefication for the insurance-question.

ZUSAMMENFASSUNG

Das klinische Bild, das zuerst von SUDECK als *»akute, trophoneurotische Knochenatrophie»* und dann von KIENBÖCK als *»akute Knochenatrophie»* beschrieben wurde und das nach ihrer Darstellung im Gegensatz zu der Inaktivitäts-Atrophie aufgestellt wird, ist ungenau und irreführend, sowohl 1) vom theoretischen Gesichtspunkt als 2) in Hinsicht auf die angewendete Nomenklatur.

1) Folgende Überlegungen drängen sich auf, wenn man das Thema *vom theoretischen Standpunkt* betrachtet:
   a) »Trophische» Nerven sind (hypothetische) *Organe*, Inaktivität ist eine *Ursache eines pathologischen Zustands. Diese zwei Begriffe können einander deshalb nicht als direkte Gegensätze gegenübergestellt werden.* Einem Verlust an Knochensubstanz durch »trophische» Nerven muss man als Antithese einen Verlust an Knochensubstanz durch andere *Organe* gegenüberstellen. Einem Verlust durch Inaktivität ist ein Verlust infolge anderer *Ursachen* entgegenzusetzen. Es liegt kein Anlass vor anzunehmen, dass die Inaktivität vermittelst anderer Organe einen Einfluss ausübt als durch diejenigen, durch welche die Krankheiten und Zustände wirken, welche die Ursache der »akuten Knochenatrophie» sein sollen.
   b) Es scheint *kein Grund vorzuliegen für die Annahme, dass »trophische» Nerven* für den als »akute Knochenatrophie» bekannten Zustand *von irgend welcher Bedeutung sein sollten.*
   c) *Die Hauptursache für das Auftreten der »akuten Knochenatrophie» ist zweifellos die Inaktivität. Gefäss- und Nervenschädigungen — sowohl toxischer als traumatischer Art — können wahrscheinlich zu einer Steigerung des Verlustes an Knochengewebe beitragen.*

2) Hinsichtlich des Terminus »akute Knochenatrophie» wäre folgendes zu bemerken:
   a) *Die Veränderungen sind nicht »akut»* im Sinne der gewöhnlichen Definition des Wortes.
   b) Die Bezeichnung »akute Knochenatrophie» ist ein Terminus, der als Parallele zu dem Namen der gleichzeitig in den Muskeln auftretenden Veränderungen der »Muskelatrophie» geprägt worden ist. Aber während »Muskelatrophie» *Verminderung des Volumens* bedeutet, ist »Knochenatrophie» eine *Veränderung in der Substanz.*

Die Veränderung im Muskel, die dieser Substanzveränderung entspricht, nennen wir *»Muskeldegeneration»* (fettige Degeneration).

Um eine zutreffende Nomenklatur zu bekommen, möchte Verf. deshalb vorschlagen, mit »Knochenatrophie» eine *Verkleinerung der Knochendimension* zu bezeichnen (konzentrische Atrophie), während *degenerative Veränderungen in der Suhstanz der Knochen* (konzentrische Atrophie) »Knochendegeneration» (oder »Lipomazia») genannt werden sollte. Der Terminus »Rarefikation» könnte als Ausdruck für Knochendegeneration im röntgenologischen Sinne verwendet werden.

Nach Beschreibung des klinischen Bildes geht der Autor auf eine kritische Prüfung der vielen divergierenden Ansichten über die Entwicklung, Ursachen und die Erklärungen der Krankheit ein. Er hält sich — auf Grund von Tierexperimenten — für berechtigt, es als Faktum aufzustellen, dass in Verbindung mit jederlei schwererer Schädigung der Extremitäten immer eine Knochensubstanzverminderung eintritt, dass diese Verminderung aber, wenn sie nur im geringeren Ausmass auftritt, im Röntgenbild nicht erkannt werden kann. Eine Rarefikation von ungefähr 10 % ist in der Regel röntgenologisch in den Knochen von Kaninchen zu erkennen.

Das fleckige Stadium ist wahrscheinlich nur bei hohen Graden von Rarefikation zu beobachten. In der Regel tritt dieses Stadium recht bald nach dem Einwirken des kausalen Faktors auf und verschwindet im Laufe weniger Wochen oder Monate, aber der Autor hat es auch noch ein Jahr nach dem Zustandekommen der Läsionen konstatieren können.

Die Tierexperimente des Verfassers zeigen *in einer Reihe von Fällen im grossen und ganzen nach verschiedenen Läsionen die eben so schnelle Entstehung und ebenso starke Entwicklung der Rarefikation wie bei reiner experimenteller Inaktivierung* (Gipsverband). Alle Versuche wurden an den unteren Extremitäten von Kaninchen ausgeführt. *Die Knochen der affizierten Extremität zeigten immer eine Abnahme an Stärke.*

Rarefikation, ob durch Inaktivität oder durch Läsionen, giebt genau das gleiche Röntgenbild.

Die bisher in der Literatur aufgestellten Theorien, welche die Natur der »akuten Knochenatrophie» erklären wollten, werden in 8 Gruppen geteilt und einer kritischen Betrachtung unterzogen. Verfasser setzt dann seine eigene Auffassung auseinander, welche er teils auf die Literatur, teils auf seine eigenen Beobachtungen und teils auf seine Experimente an Kaninchen gründet. Sie stützt sich ferner auch noch auf gewisse physiologische Beobachtungen und Theorien und gipfelt in der Hypothese, dass *lokale Prozesse, mit den Gefässen und ihren Nerven im Zusammenhang stehen, die Probleme, die sich mit diesem Thema verknüpfen, erklären können. Er nimmt ferner an, dass Einflüsse vom Zentralnervensystem oder vom Sympathikussystem von einer*

gewissen Bedeutung sein können. Diesen Einfluss denkt er sich an die *vasomotorischen Nerven* gebunden.

Unter der Rubrik »Diagnose» betont der Autor besonders die Beziehung zwischen Rarefikation und Tuberkulose.

Er ist der Meinung, dass die Rarefikation einen beträchtlichen prognostischen Wert haben dürfte und bringt nähere Belege für diese Anschauung.

Es werden einige prophylaktische und therapeutische Schlussfolgerungen gezogen, deren Haupttendenz dahin geht, zur Vorsicht bei der Reposition von Frakturen zu mahnen, die Notwendigkeit möglichster Schonung frakturierter Knochen bei Operationen zu betonen und die möglichst frühzeitige Anwendung von Massage und aktiver und passiver Übungstherapie während des klinischen Verlaufes zu empfehlen. Die Hauptsache sei jedenfalls, Extremitäten-Immobilisierung soweit als tunlich zu vermeiden oder sie wenigstens auf die kürzeste Dauer und das möglichst geringe Ausmass zu reduzieren.

Schliesslich bringt der Verfasser in einem kurzen Abschnitt einige Gesichtspunkte betreffs der Wichtigkeit der Rarefikation für die Versicherungsfrage.

RÉSUMÉ

L'image clinique donnée d'abord par Sudeck comme »*atrophie trophonévrotique aiguë des os*» et plus tard par Kienböck comme »*atrophie aiguë des os*» opposée à l'atrophie par suite d'inactivité n'est pas juste — aussi bien 1) du point de vue théorique que 2) par rapport au terme employé.

1) *En regardant la question d'un point de vue théorique* les considerations suivantes se présentent d'elles-mêmes:
   a) Les nerfs »trophiques» sont des *organes* (hypothétiques), l'inactivité est la *cause d'un état pathologique. C'est pour cette raison que ces deux idées ne peuvent être mises en opposition directe.* Il faut distinguer d'un dépérissement d'os par des nerfs »trophiques» un dépérissement d'os par d'autres *organes.* Et a un dépérissement par suite d'inactivité, il faut opposer un dépérissement dû à d'autres *causes.* Il n'y a pas lieu de croire que l'inactivité exerce une influence par d'autres organes que ceux par lesquels les maladies et les conditions que l'on suppose être la cause de »l'atrophie aiguë des os» exercent leur influence.
   b) Il n'y a pas *de raison de supposer que des nerfs »trophiques» soient d'une conséquence quelconque* dans les cas connus comme »atrophie aiguë des os».
   c) *La cause principale des cas »d'atrophie aiguë des os» est sans doute l'inactivité. En outre la lésion de vaisseaux et de nerfs — aussi bien de nature toxique que traumatique — peut probablement conduire à un dépérissement du tissu osseux.*
2) Pour ce qui concerne le terme »atrophie aiguë des os» les considérations suivantes sont à faire:
   a) *Les altérations ne sont pas »aiguës»* d'après la définition générale de ce terme.
   b) Le terme »atrophie aiguë des os» est formé en parallèle à la désignation des altérations simultanées apparaissant dans les muscles

— »atrophie musculaire». Mais tandis que par »atrophie musculaire» on désigne une *diminution de volume*, on comprend sous »atrophie des os» une *altération dans la substance* même.

L'altération dans les muscles correspondant à cette altération-là dans la substance est nommée »*dégénération de muscles*» (dégénération lipomateuse).

Pour obtenir une nomenclature précise je propose donc qu'une »atrophie des os» désigne une *diminution en volume des os* (atrophie concentrique) tandis que des *altérations dégénérescentes dans la substance des os* (atrophie excentrique) doivent être nommées »dégénération d'os» (ou »lipomacia»). Le terme de »raréfaction» peut être employé pour désigner une dégénération dans le sens de la radiologie.

Après une description clinique l'auteur fait un examen critique des différentes vues sur le développement, les causes et l'interprétation de la maladie. Se basant sur des expériences faites avec des animaux, il considère comme certifié le fait, qu'une diminution d'os a *toujours* lieu lors de graves lésions d'extrémités de n'importe quelle sorte, mais que cette diminution ne peut être constatée par la radiographie, si elle est peu avancée. Une raréfaction d'environ 10 % est, comme règle, à signaler par les rayons X dans les os des lapins.

L'état tacheté ne peut probablement être observé que lorsque la raréfaction se produit à un degré assez prononcé. Comme règle cet état apparaît assez tôt et disparaît dans l'espace de quelques semaines ou mois; l'auteur l'a toutefois encore trouvé un an après la lésion.

Les expériences que l'auteur a faites avec des animaux montrent *dans un nombre de cas pratiquement la même genèse rapide et le même développement violent de raréfaction dans les cas de diverses lésions* que dans *des expériences d'inactivité* (appareil plâtré) — toutes les expériences ont été faites aux extrémités de derrière de lapins. *Les os des extrémités atteintes montraient tous une fragilité plus prononcée que ceux de l'autre côté.*

La raréfaction par suite d'inactivité et celle par suite de lésion donnent exactement la même image Röntgen.

Les théories antérieures citées dans la littérature médicale qui prétendent expliquer la nature de »l'atrophie des os» sont divisées en 8 groupes et soumises à un examen critique. Puis l'auteur fait part de sa propre opinion basée par partie sur la littérature, par partie sur ses propres observations et sur ses expériences avec des lapins. En outre elle s'appuie sur certaines observations et théories physiologiques et culmine dans l'hypothèse que *les procés locaux en relation avec les vaisseaux et leurs nerfs peuvent expliquer les problèmes liés à cette question. En outre il suppose que des causes provenant du système nerveux central ou sympathique peuvent avoir quelque influence.* Cette influence semble être liée aux *nerfs vaso-moteurs.*

Sous le titre de »diagnose» l'auteur appuie spécialement sur la relation entre la raréfaction et la tuberculose.

L'auteur est d'avis que la raréfaction peut être d'une valeur prognostique considérable et soutient cette idée plus en détail.

Quelques conclusions prophylactiques et thérapeutiques sont faites, d'où résulte avant tout une demande de grande précaution en réduisant les fractures, la nécessité d'épargner les os fracturés autant que possible dans les opérations et la recommandation du massage avec des exercices actifs et passifs que seraient à donner aussi tôt que possible durant le traitement clinique. Le point principal est toutefois d'éviter l'immobilisation autant

que possible ou tout au moins de la réduire à la plus courte durée et au plus petit degré.

Finalement, l'auteur donne dans un court paragraphe quelques vues sur l'importance de la raréfaction pour la question d'assurance.

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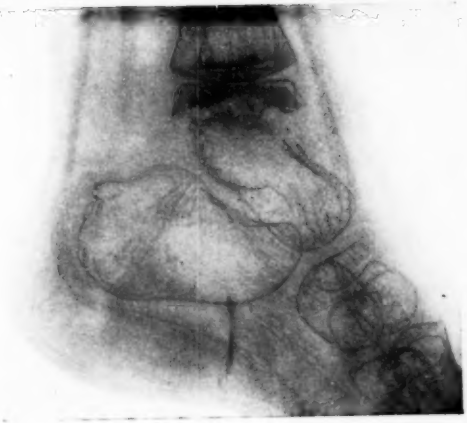
Fig. 1.



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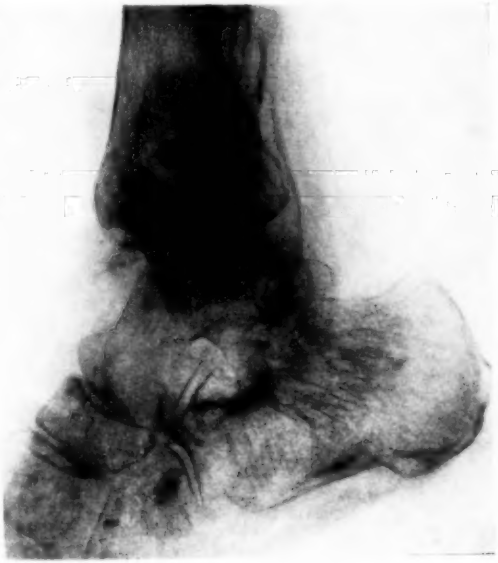

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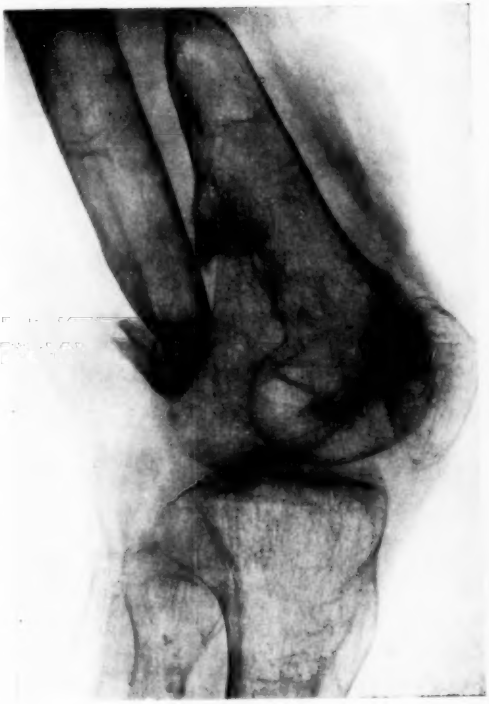

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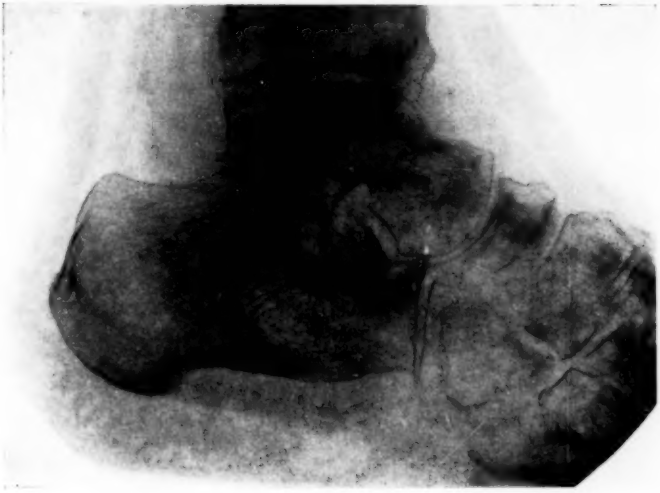

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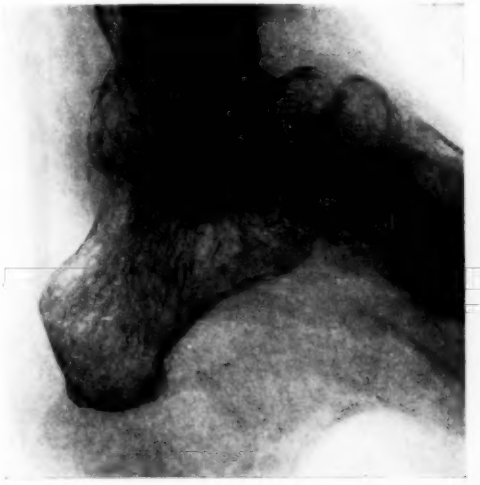

Fig. 6.

Fig. 8.

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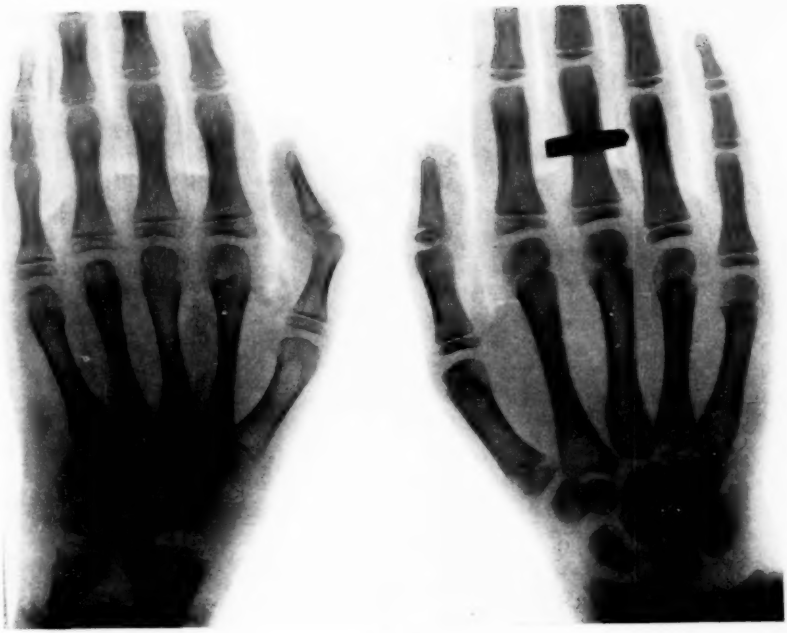

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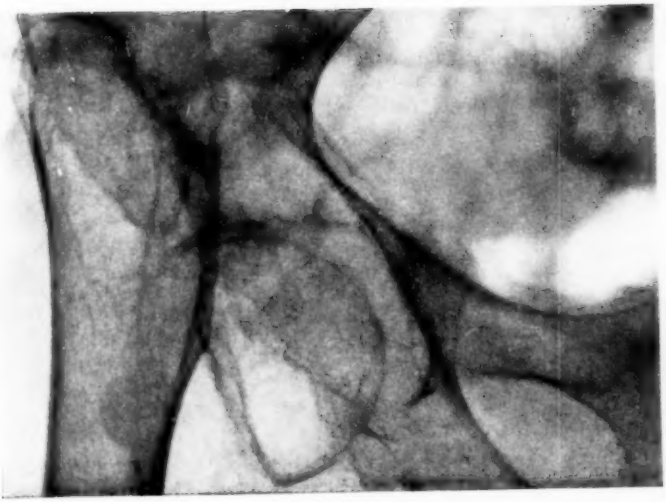

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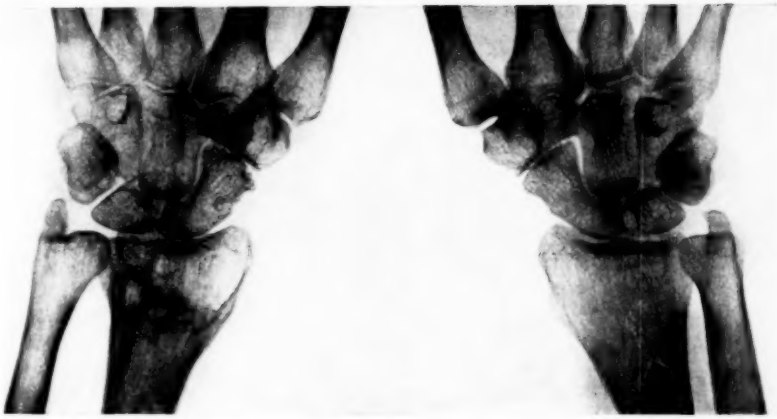

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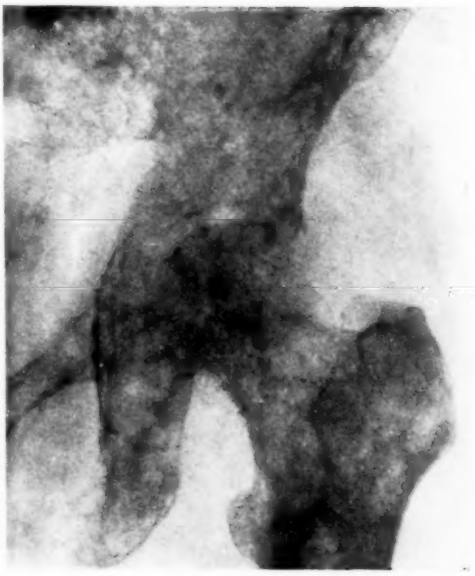

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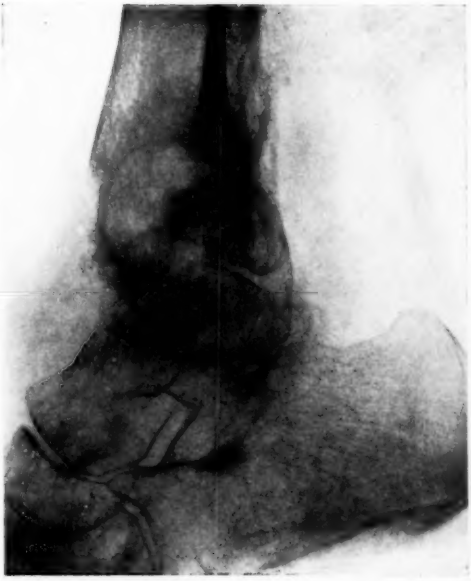

Fig. 13.

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Fortschr. = Fortschritte a. d. Geb. d. Röntgenstr.
Bruns Beitr. = Bruns Beiträge z. kl. Chir.

ROENTGEN-RAY MEASUREMENTS[1]

by

L. Baumeister,

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Translated by H. Rocky A. M. I. E. E.

A. THE DISTRIBUTION OF INTENSITY OF RAYS IN THE IRRADIATED BODY

By means of measurements on a water phantom, the reduction of intensity at the edge of the water volume has been repeatedly determined and the amount of this reduction has been ascertained. At the same time, it was further proved, that, with the increase in the field of incidence, there was also a greater difference between the intensity of the rays in the middle of the field and that at the edge of the irradiated layer of water. It must not be assumed however, that this difference, found between the medial intensity of the rays and that at the rim portions of the irradiated water cone, is to be attributed solely to the stray radiations. The stray radiations alone, are not the reason of the maximum intensity in the middle of the irradiated volume, as by the way the measurements were taken, the dispersion, that is the reduction of rays by the inverse square of the distance, must not be ignored.

Having a source of rays originating from a central spot, rays of equal intensity can only lie in an arc, respectively circular surface, and not in a straight, respectively plane surface.

The illustration (1) shows this most clearly. It is assumed that there is a focal skin distance of 30 cm, a thickness of layer of 10 cm, and a field the size of 24 cm (longitudinal side). The field is divided into 6 portions from the middle to the side (each being 2

[1] A Lecture given at the invitation of Professor Forssell at the »Radiumhemmet» (Institute) before the Members of the »Swedish Society for Medical Radiology», Stockholm, 13. IX. 22.

cm) and, starting from the focus, lines (rays) are drawn through these points. The two arcs, focal surface and focal depth, can be recognised on the illustration, and they show the focal distance is increased towards the edge of the field, if the measurements are taken in a plane surface, and in a tangent touching this surface. The increase of the focal distance from the middle of the field to the edge, is shown by the numbers on the illustration (1). We see that the focal distance of 30 cm from the middle of the field is increased towards the edge of the field to 32.3 cm.

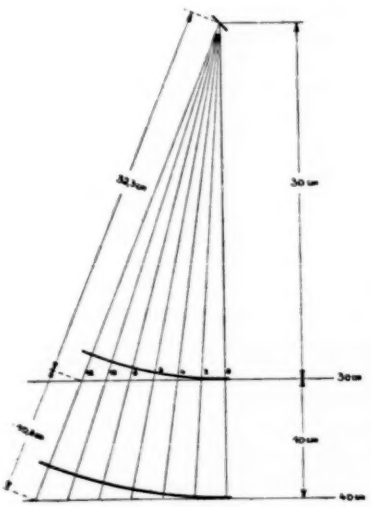


Fig. 1.

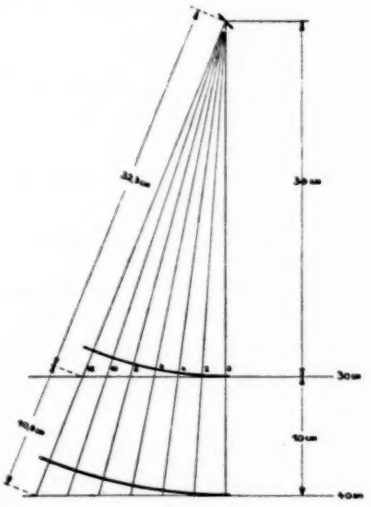


Fig. 2.

Supposing that the intensity of radiations in the middle of the field is 100 on the upper surface. The upper numerical values of the illustration (2) show how much the intensity of radiation are diminished at the edge of the field through increase of the focal distance alone.

The same applies to the depth. If at a depth of 10 cm, we have still 2 % of the upper surface intensity, then the intensity from the middle to the edge diminishes (as shown by the lower numerical values of illustration (2), more and more, until at the edge, only 17.2 % of the upper surface intensity remains. These reductions of the intensity in the depth (calculated value) are solely due to the increase of the distance (focal depth).

A further reduction of intensity of radiations in the depth 10 cm, takes place through the increase of the thickness of the layer. In the perpendicular direction, the way through a layer of equal thickness is the shortest. The further the direction deviates from the perpendicular, the longer is the way, and hence the thicker the layer. In illustration (1), the lower numerical values show how much thicker the irradiated layer of 10 cm becomes from the centre to the edge of the field. We see that the thickness of the layer at the edge becomes 10.8 cm.

The same applies to the ray filter. The rays that penetrate in a perpendicular direction (middle of the field) have the shortest way, while the rays (edge of the field) piercing obliquely through the filter have a longer way to travel.

Let us now consider all these losses in value, which arise on account of the increase of the focal distance, the increase of the thickness layer, and of the filter (on account of the oblique way of radiation towards the edges of the field) and it will be found that, with the increase of the field of incidence, a very considerable difference will be set up between the intensity of rays in the middle of the field which cannot be attributed to the stray rays.

The foregoing determinations explain why the differences that have been found between the intensity of the rays in the middle of the field and those at the edges by DESSAUER and VIERHELLER, are becoming smaller, by increased hardness of rays. The reason for this is because the hardest rays also possess the greater penetrating power and are less weakened by absorption. Consequently the reduction of intensity from the middle of the field towards the edges, (caused by the increased thickness through oblique radiation) is less with rays of greater penetration, than with rays which have a lesser penetrating power. This (increase) in the intensity of the rays at the edge of the field with very hard rays is, consequently, also not attributable to the stronger dispersion of harder rays, but instead to the slighter absorption of them.

Now the measurements of the distribution of the intensity of the rays have been carried out on the water phantom. Quite different, however, are the conditions in the case of human beings.

With the water phantom, we have a perfectly plane horizontal surface. But let us take, for example, the irradiation of the human abdomen, which is very strongly arched, with a cutaneous field of $18 \times 24 \times 30$ cm; the illustration (3) is supposed to show this diagramatically. Here half of the cutaneous field is again shown, divided into six parts (2 cm) from the middle to the edge, and, through these points lines are drawn starting out from the focus of the tube.

It is shown here that the focal skin distance from the middle of the field is considerably larger towards the edge of the field, and consequently the intensity of the rays must be considerably diminished.

The amount of diminishment of intensity on the skin towards the edge of the field owing to dispersion, is shown by the value inserted in the drawing.

The focal skin distance increases from the middle of the field (30 cm) towards the edge of the field up to 35.5 cm. If with a 30 cm focal skin distance (middle of the field), the skin dose is reached in 22 minutes, then for a 35.5 cm focal skin distance (edge of the field) a period of irradiation of 30 minutes is necessary. It must not be presumed from the foregoing, that with a large cutaneous field the intensity of the rays in the middle of the field is greater owing to the additional action of stray rays, as it is with a smaller field; it must be considered however, that the irradiation is weakened towards the edge of the field with the increase of the size of same, owing to the enlargement of the focal distance. The greater the cutaneous field selected, the more will the intensity of rays diminish towards the edge of the field.

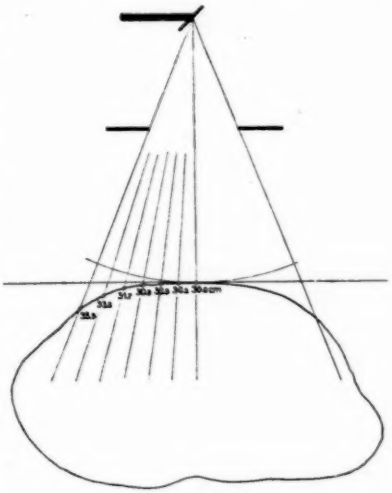


Fig. 3.

It therefore seems more suitable to irradiate with medium sized fields of incidence of 8 to 12 cm side length and with compressor tubes, because with a field of this size (see illustration 2) on the one hand there is only very slight change in the focal skin distance, and on the other, the increase of the irradiated layer of tissue towards the edge of the field, is also smaller; consequently the space irradiation is more homogenous. With tubes, which must of course posses a thin wooden bottom (like the WINTZ tubes) the cutaneous field can further be pressed out flat. With the largest WINTZ tubes, with a side length of 15 cm, recently the bottom of the tube has been shaped in the form of an arc corresponding to the focal distance for the whole cutaneous field as well as for the different depths.

If larger cutaneos fields have to be employed, then it is advisable to follow the rule practised by HOLFELDER, according to which

the focal skin distance must be at least three times as large as the side length of the cutaneous field. Thus if the cutaneous field has a lateral length of 20 cm, then the focal skin distance must be fixed at 60 cm. By following this rule, the difference between the focal middle field and the focal edge of this field is relatively small.

B. THE SUPPLEMENTARY DOSE OF STRAY RAYS

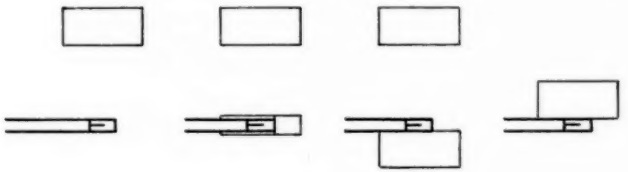
The greater the distance that the X-rays travel in a body, the deeper they penetrate, and the greater the volume that is irradiated the greater also will be the stray irradiation, and the loss of the penetrating primary Roentgen-rays, owing to absorption. This will to a certain extent be again compensated for, as the stray rays from the neighbourhood of the desired organs will reach it. This additional radiating energy is known as the Supplementary Dose.

We know that the depth dose can be improved by enlarging the field of incidence. Also by enlarging the cutaneous field. Theoretically this is due to the fact that, in consequence of the larger amount of rays, which penetrate the body through the increased cutaneous field, a correspondingly larger quantity of stray rays arises and reaches the desired organ.

The quantity of the stray rays cannot be exactly the same in all directions, as otherwise it would not be possible to improve the depth dose by increasing the cutaneous field. If the quantity of the stray rays coming from the medium, that is in the opposite direction to the primary Roentgen-rays, would be equal in quantity to those running in the same direction, then the upper surface (skin), would receive the same percentage of stray rays (supplementary dose), and a greater depth dose could not be reached. It is interesting to determine from which direction the desired organ in the depth encounters the greatest or respectively the smallest quantity of stray rays, and the following experiment was carried out for this purpose. (Illustration 4.)

Between the X-ray tube and the measuring chamber of an ionto-quantimeter, a 10 cm thick absorption layer (a wax block with a lateral length of 20 cm) was placed in such a way that the absorption layer was removed 25 cm from the mesuring chamber. The X-ray tube (COOLIDGE type) was energised by a Symmetrical Apparatus. Tube current 2.5 M. A.; potential on the tubes 200 K. V.; radiations were filtered with 0.5 mm zinc; the size of the field 6×8 cm. The time taken for the discharge of the ionto-quantimeter was 69 seconds. With this measurement, as the wax block was far

enough away from the measuring chamber, only the absorbed radiation was measured, therefore the stray rays were not measured. With the second measurement, a wax plate 20 × 20 cm was used of the same thickness as the measuring chamber, and provided with a slot into which the chamber fitted. With this arrangement the measuring chamber had wax round the sides, the back and front being left free; only laterally stray radiations were received from the wax which reached the measuring chamber as additional radiations.



Zeit	69"	60"	51"	37"
Intens.	100	115	135	186
		+15%	+35%	+86%
		17%	40%	100%

Fig. 4.

The time taken for the discharge of the ionto-quantimeter in this case was 60 seconds. This wax plate was now removed and a 10 cm thick wax block 20 × 30, arranged immediately behind the measuring chamber, so that the measuring chamber received stray rays only from the back. The time for discharge of the ionto-quantimeter, was now 51 seconds. With these three measurements, the front wax block 10 cm thick remained at a distance of 25 cm from the measuring chamber, so that the stray rays coming from the front also reached the measuring chamber. The time of discharge of the ionto-quantimeter was in this case 37 seconds. The times of discharge that have been determined in this manner give the intensity of radiations as: 100, 115, 135, 186. From this we arrive at the following: —

The stray radiation, supplementary dose, at a depth of 10 cm amounts to: —

Laterally 15 %.

Backwards 35 %.

Front in the direction of the primary rays, 86 %.

In the foregoing experiment only one wax block was ever brought

on to the measuring chamber. In the following arrangements for measuring (illustration 5) the wax blocks were removed in the following manner. In the first measurement wax blocks were placed immediately in front, by the side of, and behind the measuring chamber. In the second measurement, the wax blocks surrounding the sides of the measuring chamber were removed. In the third measurement the wax block arranged behind the measuring chamber was removed, and in the fourth measurement, the wax block arranged

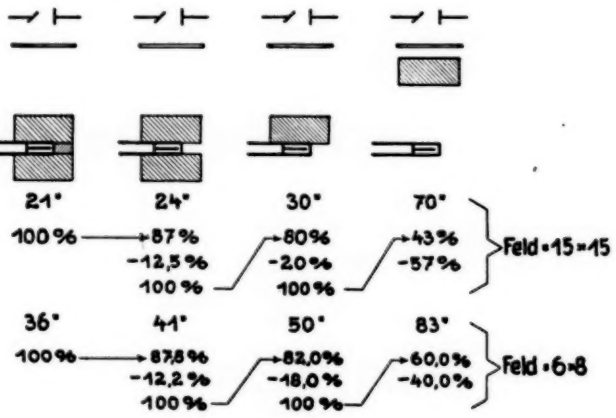


Fig 5.

in front of the measuring chamber was brought back to a distance of 25 cm. This series of measurements was gone through twice, in one case the size of the field being 15 × 15 cm; in the other with a field of 6 × 8 cm. The times of discharge that have been received, and the intensities, are shown on the illustration (5). We see again that the maximum of the stray radiation supplementary dose, reaches the desired organ or measuring chamber, from the front. Further the two series of measurements show, that the lateral supplementary dose (whether a large radiation field of incidence of 15 × 15 cm is present, or a smaller one of 6 × 8 cm), and the supplementary dose from the back differs slightly. The stray rays which reach the measuring chamber from the front, however, create a difference of 57 × 40. The lateral supplementary dose of stray rays, with the described measuring arrangement, must be the slightest, because the lateral medium in which stray rays are formed (wax block), was also the smallest. With equally large wax blocks, the minimum of stray rays takes place at the back.

The measurements for determining the stray rays reaching the desired organ (ionisation chamber or measuring chamber), require a further correction. The carrier of the measuring chamber displaces a portion of the absorption mass, from which the stray rays would also proceed. The measuring box could only receive stray rays from three sides, the fourth side was taken up by the carrier of the measuring chamber, consequently the desired organ (the measuring chamber) has received a larger amount of stray rays (supplementary dose) from the lateral direction than the one measured.

The loss which arose on account of the carrier of the measuring chamber, was determined as follows:

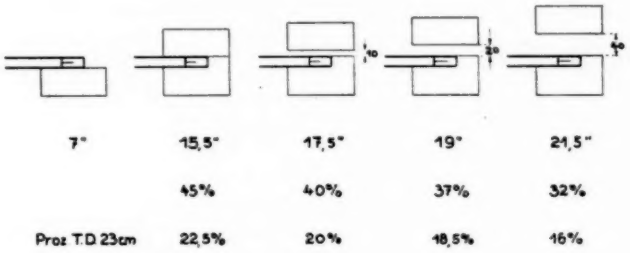


Fig. 6.

The end of the measuring chamber (front) has an addition attached to it (a metal tube) of the same dimensions as the carrier of the measuring chamber, so that now a further loss arose in the following measurement, as both front sides of the measuring chamber were now screened off. This further reduction is, therefore, just as great as that arising through the carrier of the measuring chamber. The result of measurements was that the measured lateral dose of stray rays was 8 to 10 per cent, according to the size of the field. In the measurement under consideration, it was 10 % greater, therefore 15 plus 10 % equals 16.5 %. The values from the order of measurement, according to the illustration (4) taking into account the correction, yields a diagram, as illustration (6) shows. The arrow shows the direction of the primary Roentgen-rays.

C. THE INFLUENCE OF THE DEPTH DOSE

As we have been able to see from the foregoing measurements, there is a large difference existing between the intensity of the rays, which come in contact with the measuring chamber, if, on the one

hand, the absorption body is immediately in front of the measuring chamber, and on the other hand, if there is a distance of 25 cm intervening, (with and without the stray rays, supplementary dose).

There are, however, already considerable differences present, if only relatively small intermediate air spaces exist, between the measuring chamber and those arranged in front of the absorption body. Under the same conditions, (apparatus, amount of current passing through the tube, filtration etc.) as in the foregoing measurements,

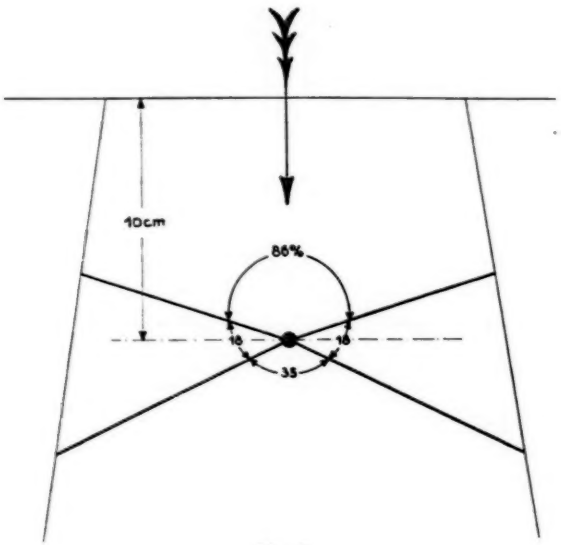


Fig. 7.

the exposed measuring chamber, with the wax blocks arranged behind it, is irradiated (illustration 7). In a second measurement, the 10 cm wax block, is placed immediately in front of the measuring chamber, and the lateral wax layer, surrounding the measuring chamber, is also placed in position. In the first case, the time required by the ionto-quantimeter to discharge was 7 seconds, in the second case 15.5 seconds. The result of this was, that the irradiation through the 10 cm of wax was reduced to 45 %. Supposing we take a 10 cm wax layer as equal to 10 cm tissue, then with a 23 cm focal skin distance we should have a 22.5 % depth dose.

$$\frac{45}{\text{Dispersion quotient}} = 22.5\ \%.$$

With three further measurements the 10 cm wax block, in front has been pushed back 10, 20 and 40 mm from the measuring chamber, so that correspondingly large intermediate air spaces arise. The times of discharge of the ionto-quantimeter were 17.5, 19 and 21.5 seconds. From this we find the percentage depth doses for a depth of 10 cm = 20, 18.5 and 16 per cent.

At the present time it is not generally recognized, that there is an ovarial carcinoma or sarcoma dose, determined by SEITZ and WINTZ, FRIEDRICH and KRÖNIG.

Now presupposing there is an ovarial dose, then the previously mentioned measurements on the phantom show how much the size of the depth dose is dependant on various circumstances. The last experiment especially, when transferred into practice, shows that an ovary lies immediately under a 10 cm layer of tissue, and can receive 22.5 % of the upper surface dose. If however, an intestinal coil with a 40 mm diameter filled with gas should be lying between the ovary and the 10 cm tissue layer, then the dose on the ovary is diminished to only 16 %. Thus although the same skin dose is given, and in both cases the layer of tissue present is of the same thickness, there may be a considerable difference present in the ovary. In the same way the deep dose can also be influenced if a layer of air should be found before or behind the ovary.

It is much easier to determine and measure the skin dose than it is the deep one, but here also there may exist different indications. The kind of trivialities, which can be easily overlooked, or passed over altogether, although they should be observed, are shown by the following example: —

In literature one comes across the opinion that the unit skin dose that has been determined for a small focal skin distance can not be utilised to determine arithmetically by means of the law of the reduction of rays, by the inverse square of the distance, for a larger focal skin distance. According to the experiences of one opinion, the time calculated must be somewhat prolonged, according to another, it must be shortened. If, therefore, the focal skin distance is not accurately measured and arranged for each individual Roentgen-ray tube, then the following may happen: —

The glass necks of Roentgen-ray tubes are of different thickness in diameter. If, therefore, with one particular tube the focal distance (skin) has been adjusted carefully to 23 cm exactly and is not again tested with another tube, it may happen that this distance is no longer 23, but instead 22 or 24 cm, according to the diameter of the neck of the Roentgen-ray tube. In the table (8), the times are registered, which are to be calculated according to the law of the in-

verse square of the reduction of rays for 50, 80 and 100 focal skin distance, if at 23 cm focal skin distance, the skin dose is reached in 40 minutes.

Let us suppose a case in which when determining the irradiation time for the skin dose, the focal skin distance was only 22 cm and not 23 cm as assumed, then the actual time would be 40 minutes with 22 cm and not 23 cm which was determined for the skin dose. Now for the larger focal skin distance, longer times would be required (table 8). Thus for example, with 100 cm focal skin distance, irradiation must be continued for 70 minutes longer. The contrary would be the case, if instead of the usual assumed focal skin distance of 23 cm, one of 24 cm were present (table 8). The skin dose was reached in 40 minutes not with 23 cm but with 24 cm. For larger focal skin distances, correspondingly shorter times are required for the skin dose (than those calculated from 23 cm). With a focal skin distance of 100 cm, irradiation must be 62 minutes shorter. The table values show what great effects small causes may give, as not only in the skin, but also in the depth correspondingly more or less is received.

F.H.	23cm	22cm
23cm	40 Min.	40 Min.
50 "	189 "	207 "
80 "	484 "	528 "
100 "	756 "	826 "

F.H.	23cm	24cm
23cm	40 Min.	40 Min.
50 "	189 "	173 "
80 "	484 "	444 "
100 "	756 "	694 "

Fig. 8.

Before all, it should be shown how very carefully one should work with deep irradiation, and that even with the greatest accuracy and skill, doses of varying size may be applied to the desired organ.

SUMMARY

The author proves, by illustrated diagrams, the errors that occur in the measurement of Dosage on account of the difference between the length of the path of the central ray, and the path of the ray at the edge of the port of entry. He further proves that, on account of this diminution of the intensity of rays at the edge of the field, it is of greater advantage to use small or medium sized ports of entry, using Compressor tubes, to reach a more uniform intensity of the rays applied, than very large fields.

His explanation of the supplementary Dose that reaches a certain organ in the depth, is also of very great interest, for the correct calculation of the effective depth Dose.

He further brought forward a number of small details which influence the Dose organs may receive in the depth, in spite of the same Unit Skin Dose being applied in every case.

These details are well worth noting by every Radiologist Practising Deep Therapy, as they clearly show how greatly the correctness of the calculated Dose depends upon the accuracy and attention to all details whith may influence the density of Rays, and the secondary rays which are generated in the surrounding tissue.

ZUSAMMENFASSUNG

Der Autor weisst an der Hand abgebildeter Diagramme die Irrtümer nach, die bei der Messung der Dosierung infolge der Differenz entstehen können, die zwischen der Weglänge des zentralen Strahles und derjenigen des Strahles an der Rändern der Eingangspforte besteht. Er zeigt ferner, dass es wegen dieser Verminderung der Instensität der Strahlen am Rande des Feldes vorteilhafter ist, an Stelle sehr grosser Felder kleine oder mittelgrosse Eingangspforten anzuwenden, indem man Kompressorröhren benützt, um eine gleichförmigere Intensität der angewendeten Strahlen zu erreichen.

Seine Erklärung der supplementären Dosis, die ein gewisses Organ in der Tiefe erreicht, ist auch für die korrekte Kalkulation der effektiven Tiefendosis von grossem Interesse.

Verfasser bringt ferner eine Reihe kleiner Details vor, welche die Dosis, die ein gewisses Organ in der Tiefe erhält, verschieden gross gestalten können, trotzdem in jedem Falle die gleiche Einheitshautdosis appliziert wurde.

Diese Details sind für jeden Radiologen, der Tiefentherapie ausübt, beachtenswert, da sie deutlich zeigen, wie sehr die Korrektheit der berechneten Dosis von der Genauigkeit und Aufmerksamkeit auf alle Einzelheiten abhängt, welche die Dichte der Strahlen und die Sekundärstrahlen, die im umgebenden Gewebe erzeugt werden, beeinflussen können.

RÈSUMÉ

L'auteur démentre par des diagrammes que des erreurs dans l'évaluation de la dose des Rayons X peuvent se produire quand en néglige de tenir compte de la différence entre la longueur des rayons centraux et périphériques frappant une surface. Il démentre en outre qu'en raison de la diminutien de l'intensité des rayons périphériques il est plus avantageux d'éclairer des surfaces de dimensions petites ou moyennes que des grandes — en utilisant des tubes compresseurs — afin d'obtenir une intensité de rayonnement plus égale.

Son exposé concernant la dose supplémantaire que reçoit un organe profond est également de grand intérêt pour le calcul exact de la dose efficace profonde.

Puis il parle d'un nombre de petits facteurs qui peuvent modifier la dose reçue par les organes profonds même quand l'unité de la dose cutanée reste la même dans les différents cas. Ces facteurs méritent d'attirer l'attention de tous les radiologues qui pratiquent la radiothérapie profonde. L'auteur démentre en effet que l'exactitude du calcul de la dose dépend largement de l'exactitude avec laquelle on évalue d'une part l'influence de tous les facteurs capables de modifier l'intensité des Rayons X et d'autre part l'influence des rayons secondaires émanant des tissus environnants.

FROM BISPEBJÆRG HOSPITAL, MEDICAL DEPARTMENT C. COPENHAGEN.
(CHIEF PHYSICIAN: K. SECHER, M. D.)

RESTORATION OF THE NORMAL AURICULAR RHYTHM IN A PATIENT, SUFFERING FROM GRAVES' DISEASE AND AURICULAR FIBRILLATION, AFTER X-RAY TREATMENT

by

Carl Schwensen, M. D.

Most authors agree that auricular fibrillation is the only abnormal rhythm which is proved graphically to be present in patients with GRAVES' disease (FRIDERICIA,[1] STRICKLAND GOODALL,[2] BRACHMANN,[3] ROMBERG.[4]

They all mention that this abnormal rhythm off and on is seen to disappear after an operation on the thyroid gland.

In his paper J. FR. FISCHER[5] describes his results of X-ray treatment of patients suffering from GRAVES' disease, but he does not say anything about the effect of the X-ray treatment on GRAVES' disease in combination with auricular fibrillation.

ROMBERG (l. c.) is the only one, as far as I have found, who mentions the result of the X-raying of patients with GRAVES' disease combined with auricular fibrillation, but he speaks distinctly against the X-ray treatment in these cases.

As the X-ray treatment of GRAVES' disease now is the routine treatment in many hospitals, I think, that the following case might be of considerable interest.

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[1] FRIDERICIA: Ugeskrift for Læger, Copenhagen. 1916, 43, 1899.

[2] STRICKLAND GOODALL: Practitioner, London. Vol. CV. No. 625. No 1. 37. (July 1920).

[3] BRACHMANN: Lancet. February 1921. 347.

[4] ROMBERG: Lehrbuch d. Krankheiten d. Herzens etc. Ed. 3. 1921. Stuttgart. 94 & 446.

[5] J. FR. FISCHER: Acta radiolog. vol. I. fasc. 2 pag. 179.

HISTORY OF THE CASE

A woman of 28 was treated as in-patient from the $^{4}/_{1}$ to $^{25}/_{3}$ 1921 for GRAVES' disease and auricular fibrillation.

She had never had rheumatic fever, chorea, diphtheria or scarlet fever. From October 1920 she felt tired and complained of shortness of breath and palpitations. There was a heavy loss in weight; her hair began falling off and she perspired profusely. There was some diarrhea. The patient had not suffered from any mental worry. Her eyes had always been a little protuding and she had not observed whether her throat had enlarged or not.

On her admittance to the hospital it was found that:

The thyroid gland was diffusely enlarged. Some exophthalmus but neither GRÄEFE'S nor MÖEBIUS' or STELLWAGS symptoms were present. Slight pulsation was seen on the neck. The skin was moist, no distinct tremor was present. On examining the heart the following was found: Ictus was felt outside the nipple line. The relative dullness extended 2 cm. to the rigth of the right sternal margin. The action was 160 pr. minute and quite tumultuary, the radialis pulse, counted simultaneously, being only 100, the pulse deficit thus being 60 pr. minute. No murmurs were heard over the precordial area. There was no stasis of the organs.

The WASSERMANN test gave a negative result; the urine was found to be normal and the blood-sugar was 0.108 (taken fasting).

She was very restless especially at night. She was kept in bed and bromide of sodium was administred. By this treatment she improved slightly, her weight being 60.6 Kg. a week after the admittance and 64.2 Kg. a fortnight later.

On the $^{24}/_{1}$ and on the $^{26}/_{1}$ she was treated with X-rays at *Bispebjærg Hospital X-ray department*. Each time 3 S. N. were administred, a 4 mm. thick aluminium filter being used. One day the right, the other day the left lobe of the thyroid gland was treated.

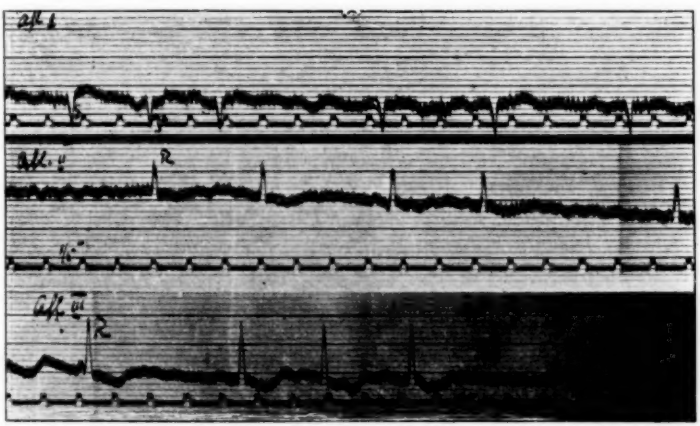

Fig. 1.

Directly after the last treatment she developed a slight fever, had a bad headache and felt unwell. Furthermore, there came a profuse perspiration. She remained like this for 5—6 weeks. During that time her weight decreased to 55 Kg. on the $^{9}/_{3}$, from which day she gained in weight, her weight on the $^{23}/_{3}$ being 57.9 Kg.

The heart remained unaltered. There was never found any signs af stasis in the organs. For some time she was treated with digitalis and iodide of potassium without any perceptible result at all.

The electrocardiogram was taken twice and showed each time auricular fibrillation with a pronounced preponderance of the right ventricle (S deepest in lead I, R highest in lead III, see fig. I).

One month after her discharge she came to be *reexamined*. She had gained 5 Kg. in weight. The struma and the exophthalmus were in the same condition as on her admittance. The dullness of the heart was now normal and electrocardiograms showed quite normal features, and especially there was now found normal P-waves and the signs of preponderance of the right ventricle had disappeared. She had not been under treatment with any medicine while at home.

Reexamination on the $^{17}/_{11}$, *1921:* She had gained enormously in weight, weighs now 80.9 Kg. She feels perfectly well. The examination of the heart showed normal features. Electrocardiograms (see fig. 2): quite normal but for a few extrasystoles, probably arising in the junctional tissue.

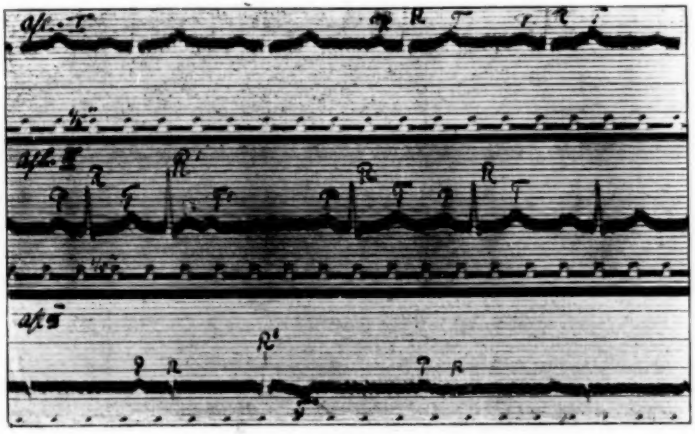

Fig. 2.

SUMMARY

In this case we have thus seen just the same result of an X-ray treatment of a patient suffering from combined auricular fibrillation and GRAVES' disease as after an operation on the thyroid gland. First a state of pronounced hyperthyroidism; after some time this seems to have stopped as the patient gains in weight and improves considerably. At the same time the auricular fibrillation has vanished and the auricular action is now found to be quite normal.

Furthermore, this case shows the very interesting fact that the electrocardiograms showed undeniable signs of a rightsided preponderance while the auricles were fibrillating, but when the auricles had returned to normal action, the normal balance between the right and left side had also been restored.

ZUSAMMENFASSUNG

In diesem Fall konnten wir nach Röntgenbehandlung einer Patientin mit Vorhofflimmern und Basedowscher Krankheit ganz dasselbe Resultat sehen, wie es nach einer Operation der Thyreoidea zu beobachten ist: Zunächst einen Zustand von ausgesprochenem Hyperthyreoidismus; nach einiger Zeit scheint derselbe aufgehört zu haben, indem die Patientin an Körpergewicht zunimmt und sich ihr Zustand beträchtlich bessert. Gleichzeitig verschwand das Vorhofflimmern und die Vorkammertätigkeit zeigt jetzt einen ganz normalen Befund.

Ferner zeigt dieser Fall die sehr interessante Tatsache, dass die Elektrokardiogramme zweifellose Anzeichen einer Präponderanz der rechten Seite aufwiesen, solange das Vorhofflimmern bestand, während nach Rückkehr der Vorkammern zu ihrer normalen Aktion auch das normale Gleichgewichtsverhalten zwischen der rechten und linken Seite wiederhergestellt war.

RÉSUMÉ

Dans le cas rapporté, un malade atteint de la maladie de GRAVES combinée à la fibrillation auriculaire, nous avons vu le traitement aux rayons X produire le même résultat que l'on observe après l'opération du corps thyroïde. D'abord un stade de hyperthyroïdisme prononcé; après quelque temps cet état paraît cesser, le malade augmente de poids et son état général s'améliore considérablement. En même temps. la fibrillation disparaît et la fonction auriculaire devient normale. De plus, on trouve chez ce malade le fait suivant très intéressant: Les électrocardiogrammes présentaient des signes indéniables d'une prépondérance du ventricule droit pendant la période de la fibrillation auriculaire, mais du môment où la fonction des oreillettes redevenait normale, l'équilibre normal entre les deux ventricules s'était également rétabli.

AUS DEM HOWARD A. KELLY HOSPITAL, BALTIMORE, MD. U. S. A.

NOCH EINMAL ZUR FRAGE DER INTENSITÄTSVERTEILUNG DER γ-STRAHLEN RADIOAKTIVER SUBSTANZEN INNERHALB EINES ABSORBIERENDEN MEDIUMS

von

Otto Glasser, Baltimore, Md.

Öffentliche Diskussionen in wissenschaftlichen Zeitschriften führen leider selten zum erfreulichen Ziel des Verständnisses; doch sei der Versuch unternommen!

Herr R. M. SIEVERT fasst in einer Betrachtung (diese Ztschr. Vol. II. Fasc. 1) meine Zeilen (d. Ztschr. Vol. I. Fasc. 4) leider als Kritik seiner wertvollen Arbeit (d. Ztschr. Vol. I. Fasc. 1) auf.

Dem aufmerksamen Leser meiner diesbezüglichen Zeilen wird nicht entgehen, dass ich keineswegs Herrn SIEVERTS Arbeit allgemein kritisiere, sondern ausdrücklich »versuche, etwaige Differenzen der unter ähnlichen Versuchsbedingungen angestellten Untersuchungen» einer Kritik zu unterziehen.

Dabei führt die Betrachtung der da »berechneten Werte» in logischer Folgerung zur Kritik und Richtigstellung *unserer*[1] [2] vor 4 Jahren veröffentlichten Werte, die mit einem »unwahrscheinlich» hohen Absorptionskoeffizienten gewonnen waren. Warum das geschah, habe ich in meiner Ausführung klargelegt! Selbstverständlich sind, der Berechnungsmethode nach, SIEVERTS Werte durchaus mit unseren vergleichbar, und es bedurfte nicht noch einmal der ausführlichen Berechnung und Darlegung der Dinge, um das zu demonstrieren.

Bei der Festlegung »gemessener Werte» möchte ich mich mit Herrn SIEVERT hüten, von »richtigen» Messanordnungen bei der Verwendung kleiner Ionisationskammern zu sprechen. Aus ausführlichen, eigenen Untersuchungen über Arbeiten und Vergleichsmessungen[3] [4] mit kleinen Kammern sind die Schwierigkeiten zu ersehen,

[1] O. GLASSER, Diss. Freiburg i. Br. 1919.
[2] W. FRIEDRICH u. O. GLASSER, Strahlentherapie. XI. S. 20. 1920.
[3] W. FRIEDRICH u. O. GLASSER, » XIV. S. 362. 1922.
[4] O. GLASSER, Amer. J. of Roentgen. a. Rad. Ther. X. S. 1. 1923.

die bei solchen Messungen und ihrer praktischen Verwertung auftreten.

Herr SIEVERT weist dann auf die Bleifilterung des Radiums in den »meisten γ-Strahlentherapie treibenden Ländern (z. B. England, Frankreich, Amerika, Skandinavien)» hin, um darzustellen, dass bei dieser dort allgemein gebräuchlichen Filterung die Absorptionsverhältnisse die von uns vor Jahren[1 2 3] beobachtete, eigentümliche Kurvenform der Äquiintensitätsflächen um eine Radiumkapsel nicht zustande kommen liessen. Das ist richtig, doch sei bemerkt, dass hier an den grössten Radiuminstituten (*Howard A. Kelly Hospital* — 5 gr. Radium Ell.; Memorial Hospital, *New York* — 4 gr. Radium El. etc) das Messingfilter durchaus verbreitet ist. Neben dem Augenschein gestattet weitgehende Literatur diese Tatsache zu verfolgen. Unsere experimentell festgelegten Verteilungskurven bestehen da also auch durchaus zu Recht, sofern das Radiumsalz selbst zur Bestrahlung verwandt wird. Dass sie zu Recht bestehen, m. a. W. dass unsere von Herrn SIEVERT bemängelte (weil zum Teil durch Extrapolation gewonnene) Kurvenform praktisch von der grössten Bedeutung ist, haben jahrelange Erfahrungen gezeigt. Literatur gibt weitgehenden Aufschluss über die auf klinischem (*Opitz*, *Schmitz*, etc), biologischem (*Jüngling*, *Beigel* etc.) und physikalischem Wege (l. c.) gefundenen Resultate.[4 5 6 7]

Wo also — nach Herrn SIEVERT — »die Unhaltbarkeit der von O. GLASSER aufgestellten Behauptungen etc.» liegt, mag der mit der Literatur vertraute Leser selbst entscheiden.

Noch einmal auf die Richtigkeit des SIEVERTschen Hinweises auf den ungeeigneten Namen »Isodosen» einzugehen, möchte ich mir hier ersparen, zumal ich mir vor Monaten erlaubte, das Herrn SIEVERT brieflich mitzuteilen, nachdem er seine erste Kritik des Namens in der »Zeitschrift für Physik» veröffentlichte. Immerhin — Folgendes sei bemerkt: Herr SIEVERT sagt auch, dass unsere Kenntisse von der eigentlichen biologischen Strahlenwirkung noch unvollkommen sind, weiter sind auch unsere physikalischen Hypothesen über diese

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[1] W. FRIEDRICH u. O. GLASSER, Strahlentherapie. XI S. 20. 1920.
[2] W. FRIEDRICH u. O. GLASSER, ZS. f. Physik 11. S. 93. 1922.
[3] O. GLASSER, J. of RAD. 1923.
[4] H. SCHMITZ and E. HUTH, Rebman New York 1922.
[5] H. SCHMITZ, Amer. J. of ROENTGEN. VIII, 6 S. 285. 1921.
» » Journ. of Rad. IV, 1 S. 10. 1923.
» » Amer. J. of Roentgen. X, 3 S. 219. 1923.
[6] E. OPITZ u. W. FRIEDRICH, Münch. Med. Wochenschr. I, 1. 1920.
E. OPITZ » » » I XIX. 1922.
[7] O. JÜNGLING u. W. BEIGEL, Strahlentherapie XIV. W. S. 423. 1922.

Wirkung m. a. W. unsere physikalischen (absoluten) Dosentheorien unvollkommen. Und zu letzteren gehört die Absorptionhypothese, auf die sich die SIEVERTsche Kritik des Namens »Isodosen» gründet.[1] Andere physikalische Dosentheorien lassen ihn wohl zu Recht bestehen.

Ein Wort noch zum Fehlen »gerade meiner Dissertation» an schwedischen Bibliotheken. Herr SIEVERT wird bei näherem Zusehen an diesen Bibliotheken so ziemlich alle Doktordissertationen der Universität *Freiburg* i. Br. seit dem Jahre 1918 vermissen. Ich erwähnte schon in meiner ersten Betrachtung, dass die Ungunst der Zeitverhältnisse daran Schuld ist, die es dem verarmten deutschen Studenten nicht erlaubte — und leider immer noch nicht erlaubt! — die Mittel für die Drucklegung seiner Arbeit aufzubringen.

ZUSAMMENFASSUNG. Es wird versucht, die von Herrn R. M. SIEVERT in seinem Artikel (*d. Ztschr. Vol. II. Fasc. 1*) für unhaltbar gehaltenen Behauptungen von O. GLASSER ihrem richtigen Sinne nach zu erklären und rechtzufertigen.

SUMMARY. An attempt has been made to explain and to justify O. GLASSERS assertions which Mr. R. M. SIEVERT has declared untenable in his article (*this Journal, Vol. II. Fasc. 1*).

RÉSUMÉ. L'auteur tâche d'expliquer et de justifier ses assertions que, dans un article antérieur (*v. ce journal Vol. II. Fasc. 1*), Mr. R. M. SIEVERT avait déclarées intenables.

ERWIDERUNG AUF DIE VORSTEHENDEN BEMERKUNGEN DES HERRN O. GLASSER

von

Rolf M. Sievert, Stockholm

Infolge Entegegenkommens der Schriftleitung wurde mir Gelegenheit gegeben, vor Drucklegung Kenntnis von den vorstehenden Bemerkungen des Herrn O. GLASSER zu nehmen.

Etwas neues zur Beleuchtung der berührten Fragen bringt uns jener Artikel nicht, weshalb ich für eine eingehende Erwiderung den Raum dieser Zeitschrift nicht weiterhin in Anspruch nehmen möchte, zumal Herr GLASSER mir ja in den meisten Punkten Recht gibt. Ich gehe gern zu, dass der Ausdruck: »Unhaltbarkeit» etwas zu kategorisch war, wie es mir auch leid tut, dass Herr GLASSER meinen Ausführungen anscheinend doch eine etwas persönliche Note abgewonnen hat, die mir wollkommen fern lag.

Stockholm, im Juni 1923.

[1] F. DESSAUER, Theod. Steinkopff. 1923.